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CONTENTS

Gingivosis	207	Calvin L. Foss, et al.
The Reattachment of Human Periodontal Tissues Following Surgical Detachment: A Clinical and Histological Study	220	Melvin L. Morris
Temporary Splinting An Adjunct in Periodontal Therapy	229	Nathan Friedman
The Dentin: Its Physical Characteristics During Curettage	232	A. B. Riffle
The Etiology of Periodontal Disease	242	Paul E. Boyle
Report of a Study on the Relationship of Personality Variables to Periodontal Conditions	248	John H. Manhold
An Evaluation of Factors Determining Prognosis in Inflammatory and Retrogressive Periodontal Disease: A Series	252	Richard William Slatten
Letter to the Editor	265	Balint Orban
Announcements	251-267	

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THE JOURNAL OF PERIODONTOLOGY

OCTOBER, NINETEEN HUNDRED FIFTY-THREE

Gingivosis*

by CALVIN L. FOSS, *San Diego, California;* HAROLD E. GRUPE, *Amarillo, Texas;* BALINT ORBAN, *Colorado Springs, Colorado*

THE term "gingivosis" was first used by Schour and Massler[1] to designate a degenerative condition of the gingiva. Their observation was based on clinical experience gained in post-war Italy where they studied gingival diseases in starving children. "In the course of investigations . . . . in post-war Naples, an unusual and severe type of gingival disease which the authors had not seen before was observed in malnourished and chronically ailing hospitalized children. For the purpose of simplification and convenience, this condition will be referred to as gingivosis. This term suggested itself because of the clinical impression that the disease was primarily of a degenerative rather than an inflammatory nature."

In the course of some histochemical investigations of cases of "desquamative gingivitis" by Engel, Ray and Orban,[2] it became evident that the most characteristic tissue change in this disease is a disturbance in the ground substance of the gingival connective tissue. This disturbance manifests itself by a "depolymerization" of the ground substance and the cementing substance with disintegration of collagenous fibers and is undoubtedly brought about by depolymerizing enzymes.

There are few histopathologic studies of this disease. Ziskin and Silvers[3] published a case in which desquamative gingivitis and lichen planus occurred simultaneously, and Ziskin and Zegarelli[4] presented twelve cases which were examined clinically and histopathologically.

In the light of these observations,[1,2] it seemed necessary to extend a new investigation of similar clinical cases and to study the tissue changes with the new observations in mind.

The present study is based on the observation of eighteen cases—sixteen women and two men—studied not only clinically, but also by microscopic examination of biopsy specimens. Not all eighteen cases will be described in detail, but those presented are typical for the entire group.

The clinical features have been described in detail by Ziskin and Zegarelli,[4]

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Fig. 1. General view of a biopsy of gingivosis (Female, age 40 (F-281)). a. Marginal gingiva; b. Attached gingiva—epithelium very irregular Inflammation in entire gingiva, marginal as well as attached.

Prinz,[5] Merritt,[6] Sorrin,[7] Hirschfeld[8] and others whose work has been presented in papers and in textbooks. Little could be added to these reports. The emphasis in the present treatise is on the histopathologic changes.

The first case is that of a healthy woman, age 40, with early signs of menopause and no other medical history. The oral mucosa was free of lesions except those of the gingiva and alveolar mucosa. The margin of the gingiva was red, the redness extending to the attached gingiva. Stippling had disappeared. A whitish membrane covering the attached gingiva was evident in some areas. This white membrane could not be removed by wiping. Subjective symptoms were: A burning sensation, pain of gingiva when touched or during mastication, bleeding without provocation. No deep pockets were found.

A biopsy was taken from a clinically typical area between the left upper first and second bicuspids. The microscopic specimen shows a dense inflammatory infiltrate in the pocket area (Fig. 1) consisting mainly of lymphocytes and plasma cells. Polymorphonuclear leukocytes are numerous in the epithelium and immediately below it in capillaries as well as in the tissues. The inflammatory cell accumulation is not confined to the pocket area, but large masses of inflammatory cells are seen all along the epithelium of the attached gingiva, extending into the alveolar mucosa.

The epithelium is irregular. Thin areas without epithelial ridges alternate with regions where the epithelium is thick, forming heavy ridges. The connective tissue papillae are accordingly thin or wide, or completely missing. The surface of the epithelium is not keratinized. Some shedding has taken place, but the shedding cells are vital cells with nuclei. No granular layer is present (Fig. 2).

Degenerative changes in the epithelium are of greatest significance. Some cells appear normal; others are dark, picnotic. Intracellular vacuoles are frequent, and some peculiar, whorl-shaped arrangements of cells can be observed (Fig. 3). Edema and "ballooning" of cells in this region are in evidence. As can also be clearly seen in this illustration, the border between epithelium and connective tissue becomes indistinct, difficult to recognize. Lymphocytes migrate into the epithelium and no basement membrane is present. The epithelial cells become separated by accumulation of intercellular fluid, edema.

The epithelial cells of the prickle cell layer show extensive indication of degeneration. Hydropic changes, cloudy swelling, and different degrees of hyalinization can be observed almost any place in the epithelium, being more intense in some areas than in others. In some regions, the epithelium seems to disintegrate into a granular mass (Fig. 4); the intercellular bridges are broken up; the outline of cells

Fig. 2. Higher magnification of epithelium, showing signs of granular degeneration. There is no keratin on surface. Epithelium thin, ridges irregular.

Fig. 3. Whorl-shaped arrangement of epithelial cells. Degeneration of epithelial cells. Edema.

becomes indistinct, and the cells elongate. Present in the cells is an eosinophilic material which sometimes becomes arranged in large oval or round masses or in clumps (Fig. 5), lying close to the surface. In this region, large spaces often appear between the epithelial cells, filled with hyalin bodies or numerous leukocytes (Fig. 2).

The connective tissue is similarly affected. The ground substance seems to have undergone degeneration (depolymerization[2]) and presents a leached-out appearance (Fig. 6). Hyalin masses are present in the epithelium as well as in the connective tissue (Fig. 7). The collagenous fibrous tissue which is a characteristic structure of the gingiva, is largely missing, being replaced by the extensive edema and inflammatory infiltrate. The inflammatory cell accumulation consists mainly of lymphocytes, plasma cells and macrophages. Histiocytic elements and giant cells are a frequent finding (Fig. 8).

The second case is that of a woman 54 years of age, a hay fever victim. The onset of the disturbance had occurred a year previously. The patient complained that her gums bled easily, of a slight burning sensation and an itchy, tingling feeling of the tongue.

The gingiva appeared red; the margin normal; the papillae slightly blunted. The attached gingiva appeared glossy, the stippling having completely disappeared. Neither pockets nor exudate were present. The inside of the lip was denuded in one area, and covered with a whitish membrane.

A differential blood count revealed no pathologic features. The monocyte count was 8%, which is the upper border of normal. Two biopsy specimens, taken several months apart, showed the same pathologic conditions.

The entire gingiva—pocket side, margin, and attached region—is densely infiltrated

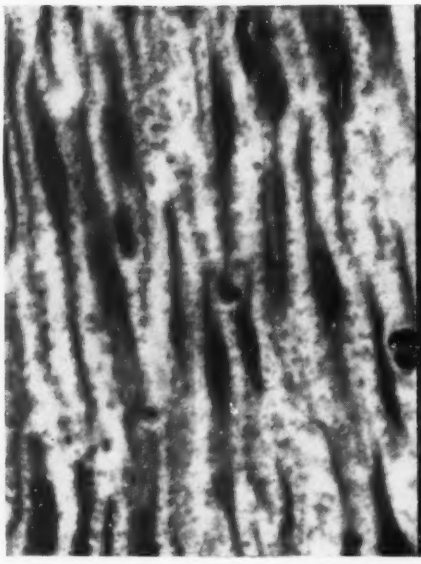

Fig. 4. High magnification of epithelium of Figure 1. Epithelial cells elongated; cytoplasm degenerated; intercellular substance forming a granular mass (eosinophilic in the H & E specimen); intercellular bridges fragmented.

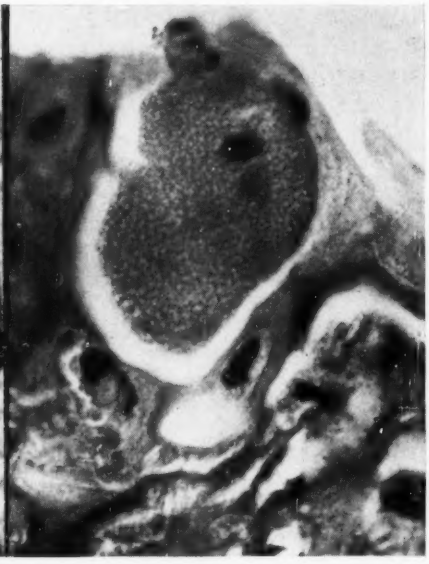

Fig. 5. Confluence of hyalin substance, forming large mass (eosinophilic in the H & E specimen).

by inflammatory cells (Fig. 9); whereas the epithelium at the margin is of rather normal thickness. The epithelium covering the attached gingiva is extremely thin. No connective tissue papillae are present; consequently the epithelium is almost evenly thin without any epithelial ridges. Only small epithelial projections are seen here and there (Fig. 10), and the surface is not keratinized.

The subepithelial connective tissue is densely infiltrated by inflammatory elements, mainly lymphocytes and plasma cells, but histiocytic elements are rather numerous (Fig. 11). These histiocytes are large cells with pale nuclei, sometimes wrinkled and irregular.

Very little connective tissue is present. The ground substance as well as the collagenous fibrous material is missing (Fig. 12). The tissue is extremely edematous, and the connective tissue ground substance appears as a fine, granular mass. The border between epithelium and connective tissue is barely recognizable, and a few irregular mitotic figures can be seen in the basal cell layer of the epithelium.

There is a peculiar tissue change around the capillaries—a heavy hyalin layer surrounds many of the otherwise thin-walled vessels (Fig. 13).

The third case to be discussed is that of a woman 43 years of age. Menopausal disturbances had been present for two years, and the patient had received estrogenic hormone treatment from her physician. Subjectively, the oral discomfort was quite extensive—the entire gingiva being painful with a burning sensation. The gingiva bled after meals or brushing.

Biopsy specimens were taken at four different periods. In this case, taking a biopsy was extremely difficult. The surface epithelium was very friable. In fact, air

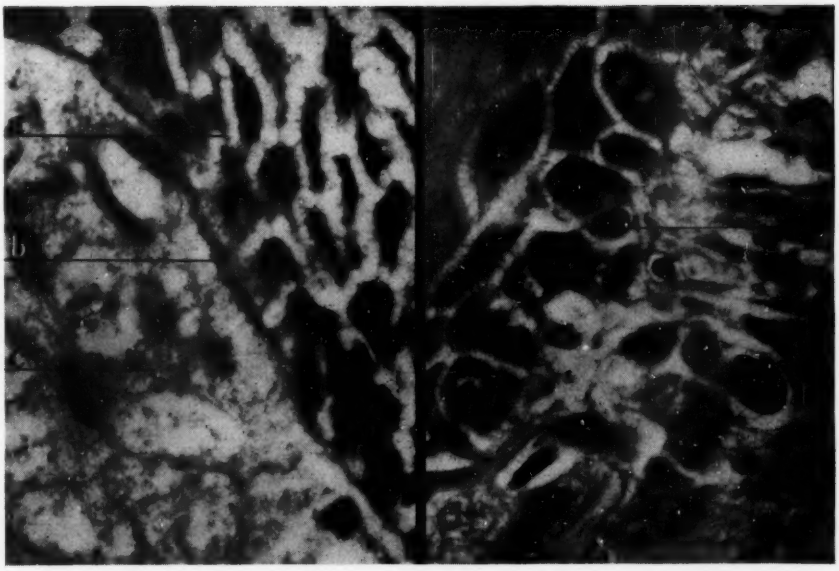

Fig. 6. Borderline of epithelium (a) and connective tissue (c), showing basement membrane (b). Ground substance of connective tissue edematous, depolymerized. No collagenous fibers present.

Fig. 7. Hyalin masses in the epithelium (a) and connective tissue (b) as an indication of tissue degeneration.

pressure upon the surface (from a chip blower) lifted the epithelium up and a large blister was formed. When one biopsy was taken between the lower cuspid and first bicuspid, a large part of the gingival epithelium (including attached gingiva) came off as a membrane all along the lower anterior region. The surface beneath was highly red, raw, hemmorrhagic, extremely sensitive. The surface was epithelialized in about four days.

One of the biopsy specimens (Fig. 14) reveals an extremely dense, inflammatory infiltrate in the entire gingiva. No connective tissue fibers are seen; the epithelial ridges are completely missing. The epithelium in two areas is lifted from the underlying, inflamed connective tissue, and bullae have resulted. One of these bullae at the tip of the gingival margin is seen in Figure 15. The epithelium is extremely thin, infiltrated by large masses of polymorphonuclear leukocytes. The fluid in this blister is a fibrinous material, also infiltrated by leukocytes. The blister developed between epithelium and connective tissue. There is no keratinization of the surface epithelium. The connective tissue is highly hyperemic and infiltrated by inflammatory cells. The inflammatory cells are mainly lymphocytes and plasma cells, but polymorphonuclear leukocytes are present close to the blister. In the deeper connective tissue, a large number of eosinophile leukocytes are present. In viewing the section of Figure 14, one can imagine that it would have taken a very short time for the blister to extend from the margin toward the attached gingiva and that it would have taken little effort to mechanically wipe off the epithelium from the underlying, severely inflamed connective tissue.

Another biopsy from the same patient reveals a somewhat different picture. Again, a bulla has formed (Fig. 16), but this bulla seems to have developed as an intraepithelial

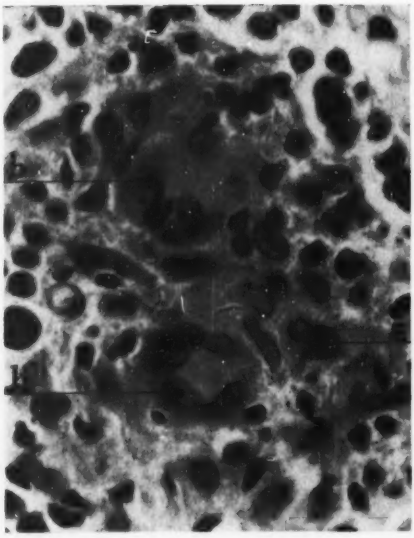

Fig. 8. Histiocytes (a) and histiocytic giant cells (b) in the connective tissue (High magnification of Fig. 1).

blister by degeneration of the epithelial cells of the prickle cell layer. The degeneration seems to be what is known in pathology as a ballooning degeneration of the epithelium. It is most frequently found in lesions produced by a virus infection such as herpes, especially herpes zoster. In this ballooning degeneration, the epithelial cells seem to separate (Fig. 17), to become round and blown up, and to float in the fluid of the blister. The blister fluid comes from the inflamed underlying connective tissue.

In another biopsy of this patient, still a different finding was obtained. The epithelium shows an extremely rare disturbance—the formation of multi-nucleated cells, giant cells, due to amitotic mitosis (Fig. 18, A and B). Such multi-nucleated epithelial cells are also found in virus infections of the skin. The possibility has to be considered in this case that a secondary virus infection has complicated the primary degenerative disease of the gingiva.

Two years after this patient was first seen, she complained of a lump in her breast. Breast cancer was diagnosed and surgically removed, but the patient died within six months after the operation.

In some cases, as shown in the fifth case (Fig. 19), the gingival margin may show the usual chronic marginal gingivitis without the characteristic features of the degenerative process. Toward the attached gingiva, however, the epithelium becomes thin; the connective tissue papillae disappear; all connective tissue seems to disappear and to be replaced by an extensive inflammatory infiltrate. The inflammatory infiltrate will often show extensive degen-

Fig. 9. General view of a gingival biopsy of gingivosis (Female, age 54 (F-171)). a. Marginal gingiva. Inflammatory reaction in connective tissue and epithelial reaction as seen generally in gingivitis; b. Attached gingiva—epithelium thin, no keratinization. Connective tissue hyperemic, edematous, densely infiltrated by inflammatory cells.

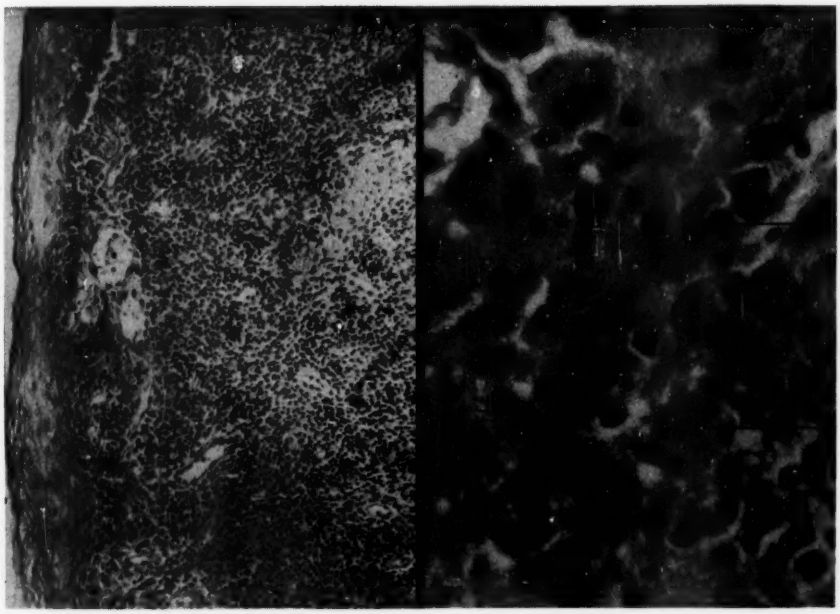

Fig. 10. Higher magnification of epithelial-connective tissue border, showing thin epithelium, no keratinization. Dilated vessels below the epithelium. Dense infiltration of inflammatory cells, large number of which are histiocytic.

Fig. 11. Histiocytic macrophages (a) from Figure 10.

eration and tissue necrosis (Fig. 20) as first reported by Ray and Orban[9]. Also, an important feature of gingivosis is that the depth of the pocket and the local irritation which may arise, have no direct relation to the severity of the inflammation in the rest of the gingiva, especially in the attached gingiva (Fig. 21). In gingivosis, the pocket is usually shallow and the inflammation does not correspond to the irritation arising in the pocket, either in extent or localization. As seen in the last two specimens, the epithelium becomes thin in the attached part of the gingiva, whereas it shows the usual signs of reaction to inflammation in the pocket area. The characteristic tissue change is loss of connective tissue ground substance (Fig. 22), accompanied by accumulation of edema and inflammatory infiltrate.

DISCUSSION

The most important finding seems to be the degeneration and breakdown of connective tissue and epithelium. The connective tissue ground substance appears to be destroyed, depolymerized, according to the findings of Engel, Ray and Orban.[2] This tissue appears in microscopic specimens as "empty"; the fibrous tissue is often fragmented and appears as a hyalin granular material. Such a granular, hyalin mass also appears in the epithelium; the intercellular spaces become wide and seem to contain a granular mass; the cells also become hydropic and a cloudy swelling and hyalin degeneration occur. The basement membrane disintegrates, blisters; bullae develop and the epithelium may be easily removed. This fact is responsible for the term "desquamative" gingivitis as used by early investigators. However, this is not a des-

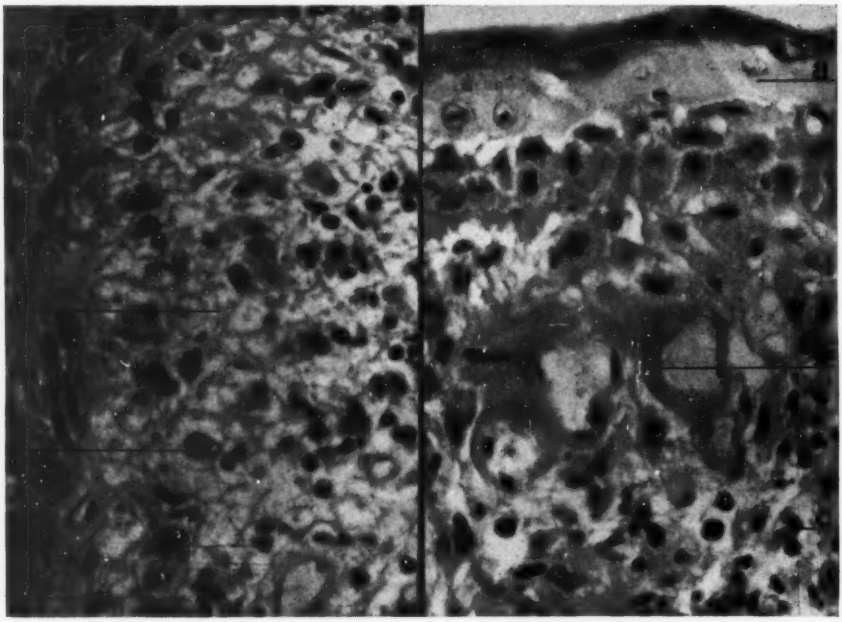

Fig. 12. Epithelial (a) and connective tissue (b) border from Figure 9. The basement membrane is vague, borderline not distinct. Connective tissue edematous, loose, lacking in fibers, infiltrated by inflammatory cells, a large number of which are histiocytes (c).

Fig. 13. Higher magnification of Figure 9, showing epithelial degeneration (a) and hyalinization of blood vessel wall (b). Histiocytic macrophages (c) in connective tissue.

quamative process; this is not excessive shedding; this is a denudation of the connective tissue by separation of the entire epithelium from the connective tissue base. The reason for the separation seems to lie in the degenerative process of the connective tissue ground substance.

While it is difficult for many to realize that this process is primarily a degenerative rather than an inflammatory process because of the ever-present severe inflammatory infiltrate, one has to keep in mind that the inflammatory reaction can be easily explained by secondary infection of the degenerated tissues. The fact that one can frequently differentiate between the marginal inflammation caused by irritation from the pocket and the inflammation in the region of the attached gingiva is, in itself, an indication that two different processes are at play. Most cases of gingivosis present no deep pockets at all (in some cases gingivosis occurs in combination with periodontitis), and it would be difficult to explain how an inflammatory process resulting from a local irritation could extend into the attached gingiva and sometimes into the alveolar mucosa. The microscopic appearance of the epithelium in this region is also an indication of a special process. The epithelium becomes thin; the epithelial ridges disappear because of the lack of connective tissue fibers; no papillary layer is present and keratinization is usually absent.

The inflammatory infiltrate is also unusual. The finding of histiocytic elements in a large number of cases is rather peculiar. Histiocytes in the gingiva have been found in eosinophilic granuloma,[10] monocytic leukemia[11] of the Schilling type, and in four of the eighteen gingivosis cases. In

several cases, extensive eosinophile leukocyte infiltration was found, in very unusual intensity, as not seen in gingival inflammation otherwise. The presence of both histiocytes and eosinophile leukocytes could be interpreted as results of tissue breakdown. Tissue breakdown (as well as the extensive tissue necrosis) is seen in the extensive degenerative process going on in the connective tissue.

The third case which was presented has shown some peculiarities, such as formation of bullae. This has been described previously by Ziskin and Zegarelli[4] in a number of cases. However, one of the blisters in this case closely resembled a herpetic lesion, although no inclusion bodies could be detected. It is, never-the-less, possible that a secondary virus infection was superimposed upon the degenerative changes characteristic of gingivosis. The severe inflammatory reaction can be explained by possible bacterial invasion or penetration of toxins through a thin, degenerated epithelium which is not keratinized. The fact that the attached gingiva is the most frequently and severely involved, might find its explanation in the fact that under normal conditions, this part of the gingiva is extremely rich in collagenous, fibrous connective tissue. It is conceivable that areas rich in such tissue and exposed constantly to local irritating factors, mechanical as well as bacterial, are the first to undergo pathologic changes of depolymerization with certain systemic disturbances.

Most of our cases were of women at the age of menopause. There were only two men in the eighteen cases. These two men both were in the fifties age group. One was

Fig. 14. Bullous lesion (a) of gingiva in gingivosis (Female, age 43—(G-34)). The epithelium is thin, shows no ridge formation and is elevated from connective tissue by fluid accumulation (b). Connective tissue densely infiltrated.

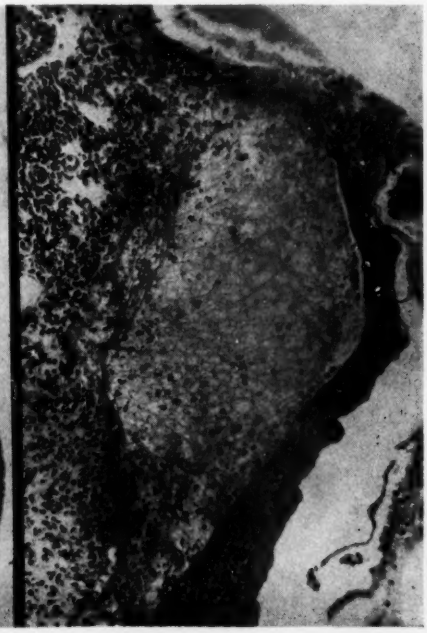

Fig. 15. Higher magnification of marginal bulla of Fig. 14. Bulla is located between epithelium and connective tissue. Bulla is filled with fibrinous exudate and leukocytes. Acute inflammation at the connective tissue border.

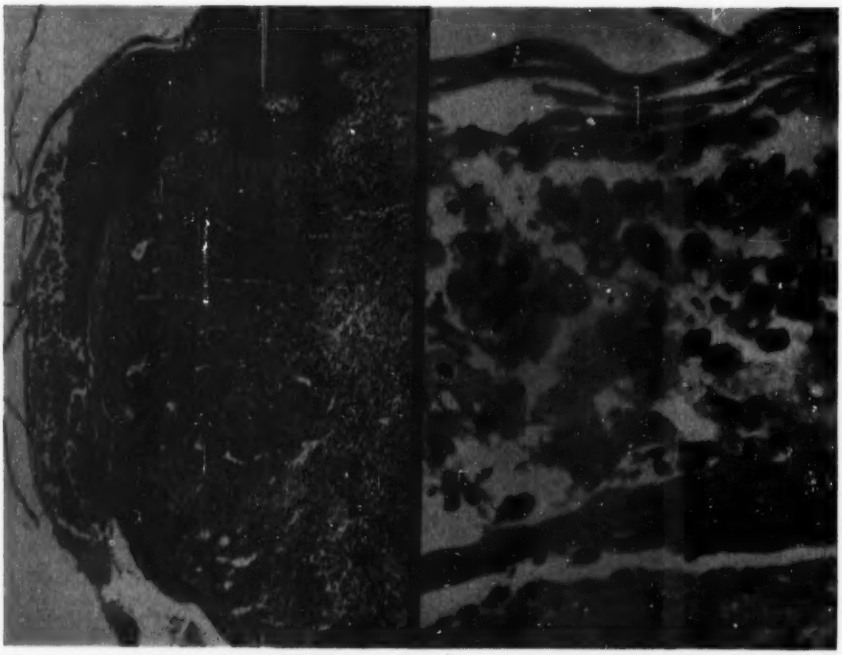

Fig. 16

Fig. 17

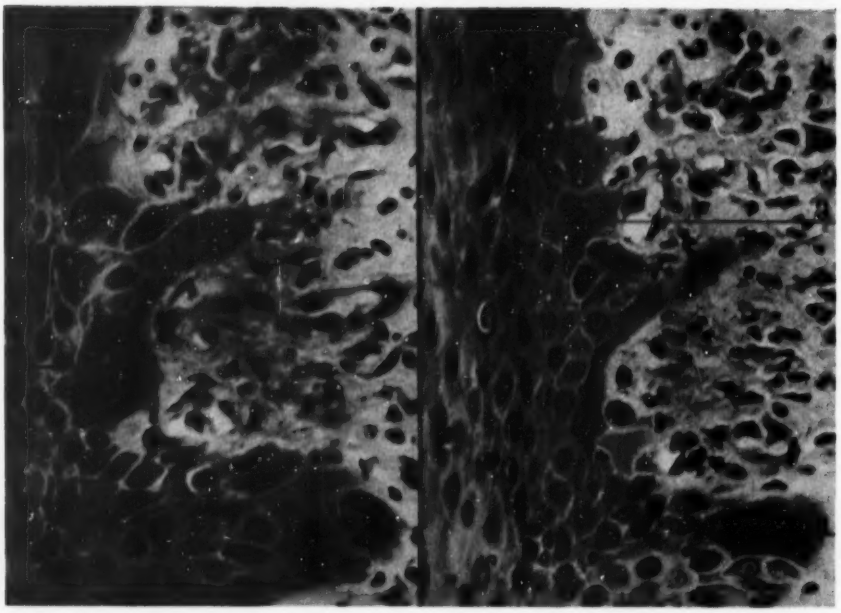

Fig. 18(A)

Fig. 18(B)

Fig. 19

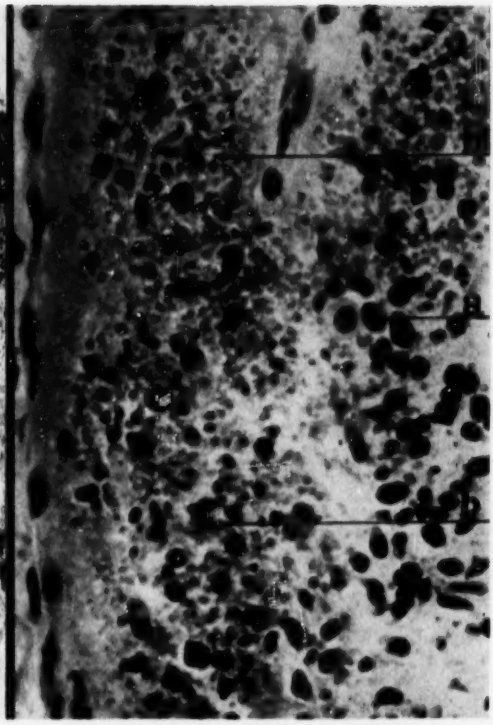

Fig. 20

Fig. 19. Gingival biopsy of gingivosis—(Male, age 46 (G-400)). Marginal gingiva (a) shows usual picture of gingivitis, originating in pocket area (b). Attached gingiva (c) shows severe degenerative signs, inflammatory infiltrate of plasma cells, necrosis in connective tissue, and extremely thin, unkeratinized epithelium. Fig. 20. High magnification of connective tissue of Figure 19. Plasma cells (a) and masses of necrotic cells and tissue (b).

an extremely sensitive and nervous type of individual who went into a severe shock from two drops of procaine used for taking the biopsy specimen. The other male was of rather calm nature, but his complaints started after a depressing personal experience.

Fig. 16. Bullous lesion in gingivosis (same case as Fig. 14. Bulla is formed by intraepithelial degeneration (a).

Fig. 17. Higher magnification of epithelial degeneration from Fig. 16—(a) Surface epithelium; (b) "Ballooning" degeneration of epithelium; (c) Basal part of epithelium; (d) Connective tissue.

Figs. 18 A & B. High magnification of epithelium of gingiva from same case as Figs. 14 and 16. Multinucleated, giant epithelial cells (a) in the basal cell layer.

It is likely that some systemic disturbance in the endocrine function of the sex glands is the trigger factor in development of gingivosis. Ziskin recommended and successfully applied estrogenic hormones locally to the affected tissues. The locally-applied hormones had some systemic effect. However, hormones administered systemically were not effective on the diseased gingiva. Unfortunately, when the hormone was not used continuously, the disease symptoms recurred.

In several of our cases, cortisone was used locally. The rationale in this treatment was that the inflammatory reaction is a secondary feature, complicating the pri-

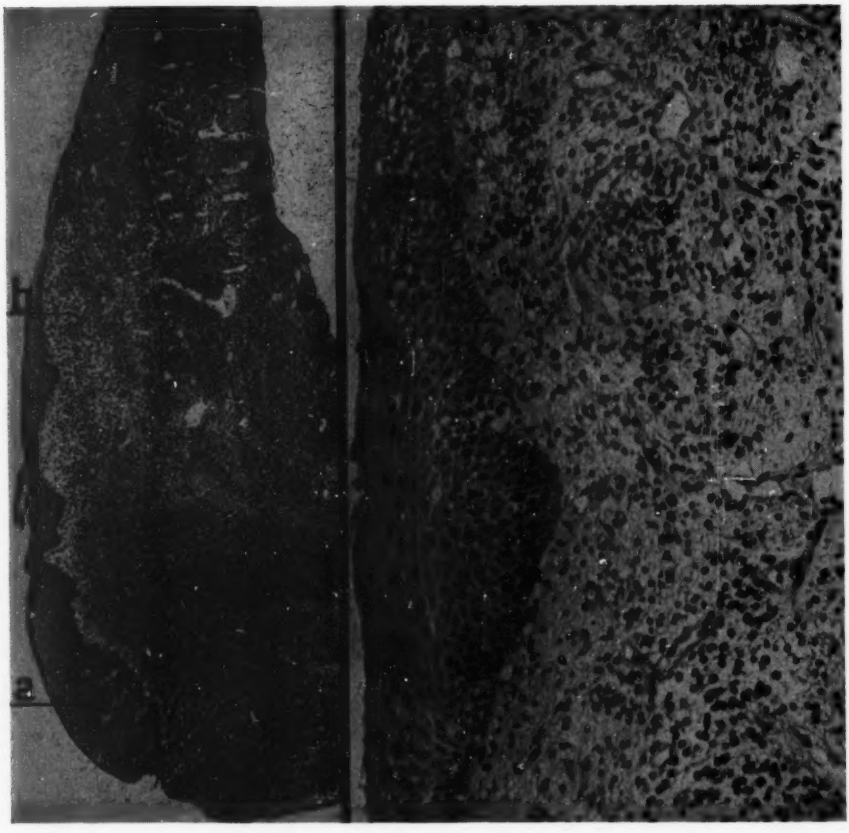

Fig. 21. Gingival biopsy of gingivosis —(Female, Age 44—(G-404). Marginal gingiva (a) shows usual signs of gingivitis. Attached gingiva (b) edematous infiltrated by inflammatory cells. Epithelium thin.

Fig. 22. Higher magnification of Fig. 21. Epithelium thin, non-keratnized, edematous. Connective tissue "depolymerized." Ground substance is edematous, shows no collagenous fibers. Inflammatory cells mainly lymphocytes and plasma cells.

mary degenerative process, preventing a healing tendency. It was observed that during application, the subjective symptoms improved. The objective symptoms showed improvement, too. In one case, the disturbance cleared up and has not returned to date (two years). However, in two other cases, after the local application of cortisone had been stopped, the objective symptoms recurred, followed later by recurrence of the subjective complaints. In one of the cases, the patient complained of considerable soreness during eating. This complaint entirely subsided with application of the cortisone, but the clinical appearance did not improve. The subjective improvement lasted for several weeks after application of the cortisone. No satisfactory explanation can be offered for these results. They are given here for what they are worth. Similar observations have been made in medicine in treating different diseases with-

out satisfactory explanation of the action of cortisone.

Diagnosis of "gingivosis" seldom causes any difficulties. However, atypical cases of this disease and an atypical lichen planus may be confused. Lichen planus seldom causes any subjective symptoms. Microscopically, it shows hyperkeratosis, whereas in gingivosis, there seldom is hyperkeratosis. The inflammatory infiltrate in gingivosis shows plasma cells, whereas plasma cells are absent in lichen planus. Gingivosis seldom extends into edentulous areas or into the cheeks or palate and tongue, whereas lichen planus is rather rare on the gingiva. Gingivosis appears with extensive redness, edema, whereas lichen planus shows the lacy whiteness of the tissues, often without inflammatory halo. Hyalin bodies appear in both conditions in the epithelium as well as in the connective tissue; however, it seems to be more massive in lichen planus.

SUMMARY

In summary, we can state that gingivosis is a degenerative disease of the gingiva. It is characterized by degeneration of the connective tissue ground substance (depolymerization), leading also to disappearance of connective tissue fibers. The papillary layer of the lamina propria disappears, and the entire lamina propria is infiltrated by lymphocytes, plasma cells, and frequently by histiocytic elements and eosinophile leukocytes. The epithelium becomes thin, showing different signs of degeneration; the epithelial ridges disappear; keratinization is rare. Because of degeneration of connective tissue (basement membrane) as well as of epithelium, the epithelium often separates from its base, the lamina propria.

The disease is probably brought about by a disturbance of the endocrine function of the sex glands.

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Beg your pardon! Attention has been called to a minor error which occurred in the article by Dr. Frank G. Everett in the July issue of the JOURNAL OF PERIODONTOLOGY. In the second paragraph, line 4, it should read "advanced periodontitis simplex" instead of "advanced periodontal simplex."

The Reattachment of Human Periodontal Tissues Following Surgical Detachment: A Clinical and Histological Study

by MELVIN L. MORRIS, B.S., M.A., D.D.S.,* *New York, N. Y.*

THE regeneration of supporting structures in the treatment of periodontal disease has been the topic of frequent discussion in dental literature. Despite reports of successful reattachment of pathologic pockets, no technique is presently available to the profession producing high percentages of success.[20] This desired goal awaits further understanding of the physiology and pathology of the periodontium.

It has been shown that normal periodontal repair readily occurs in animals in regions not associated with a gingival crevice.[3,4,5,6,7,8] Similar evidence in humans has been reported following endodontic therapy,[15] root fractures,[16] and surgical intervention through a semi-lunar flap.[7]

Observations on the sequellae of surgically created pockets have been made by a number of investigators. Skillen and Lundquist[24] examined surgically created pockets in dogs and noted that connective tissue reattachment occurred only for a few millimeters in the deepest sections. Glickman and Lazansky[13] reported complete healing in dogs following gingivectomy and flap operations. Linghorne and O'Connell[18] created surgical pockets in dogs and reported a connective tissue union with formation of new cementum on both dentin and cementum. The connective tissue fibers ran parallel to the root, in contrast to the usual direction of periodontal fibers. The greatest thickness of cementum was observed in the apical end of the wound. Similar experiments by Waerhaug[28] on dogs showed complete reattachment. Stones[25] created surgical pockets in monkeys, removing cementum in some cases. Where cementum was removed, epithelium invariably grew past denuded dentin and attached itself to the first cementum encountered. It was noted that the healed tissues tore easily in sectioning.

Workman[28] reflected surgical flaps in humans and removed the teeth with attached tissues after four weeks. Two sections showed a completely normal connective tissue attachment. Borden[10] reported a similar experiment but only presented sections from dogs. Waerhaug's human section[28] showed complete healing eight months after creation of a surgical pocket.

Ramfjord,[27] following the technique of Swenson,[26] produced pathological pockets in monkeys and removed the epithelial lining with 75% phenol, 25% camphor, and curettage.[1] Epithelial and normal connective tissue attachments were both recorded.

In humans, Box and McCall[11,12,19] have shown epithelial and parallel connective tissue fiber healing of pathological pockets. Beube[9] presented a case with an apparent connective tissue union. None of the human work (except Waerhaug's) contained landmarks in the sections indicating original pocket depths, an important criterion suggested by Kronfeld.[17]

The paucity and inconclusive nature of human histological material suggested the need for further study of periodontal pocket healing. The surgically created pocket presents simplified conditions, without the chronic inflammation or epithelial lining of the pathological variety. It was felt that more detailed knowledge of the surgical type might yield information of possible clinical value.

CLINICAL PROCEDURE

The study was conducted on periodon-

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Case No.	Tooth	OPERATION	Days of healing	Gingival recession	Surgical detachment	Clinical reattachment	REMARKS
1	1/	Simple surgical detachment	21	1	2	1	Tissues partly torn in extractions
2	/3	Simple surgical detachment	21	0	2.5	4	Tissues partly torn in extractions
3	/3	Simple surgical detachment	63	0	3	3.5	Tissues partly torn in extractions
4	/3	Simple surgical detachment	21	1	5.5	4.5	
5	3/	Cementum removed	42	1	3.5	1.5	
6	3/	Cementum removed	..	.5	4.5	0	
7	/3	Simple surgical pocket, gingival cuff removed	56	..	7	5	
13	/1	Cementum curetted	83	..	5.5	6.5	
17	/1	Cementum curetted gingival cuff removed	71	..	5	4.5	
18	1/	Coronal half cementum curetted apical half removed. Gingival cuff removed	106	..	5	4	
19	1/	Coronal half cementum removed, apical half curetted	85	1	5	4	

CHART I

Millimeter Measurements

tally sound vital upper anterior teeth condemned as useless, for prosthetic reasons. Patients were healthy adults, ranging in age from 37 to 74 years. Labial surfaces devoid of clinical inflammation were chosen for ease of access, and the relative frailness or total absence of overlying bone. These factors lent themselves to simplicity of procedure, minimal patient discomfort, and absence of deformity following extraction with adjoining tissues.

Surgical pockets, 2-3 millimeters wide, were created with a narrow scalpel. In some instances, thin labial bony plates were separated from the roots with Wiedelstadt chisels. Nicks were made with a No. 2 wheel bur, as landmarks, on the labial enamel and on the roots at the depths of the surgical pockets. Environmental conditions were varied by curetting roots, leaving them untouched, or removing cementum with a No. 2 round bur. Sutures were drawn through the adjacent papillae, encircling the teeth, holding tissue against root. In four cases the free gingival cuff was removed to delay possible epithelial invagination. The pockets were then protected with gauze for ten minutes to aid clot formation.

Fig. 1. 56-day specimen of simple surgical pocket. Connective tissue fibers (CT) parallel to root. New cellular cementum (NC) heaviest in nick (N).

Measurements were made from the enamel nick to the point of soft tissue attachment before and after surgical separation and several weeks postoperatively. In seven cases the positions of the gingival margins were similarly recorded.

Twenty cases were operated on. Eleven returned for postoperative measurements (chart I). Five biopsies were obtained, after healing intervals ranging from 21 to 106 days.

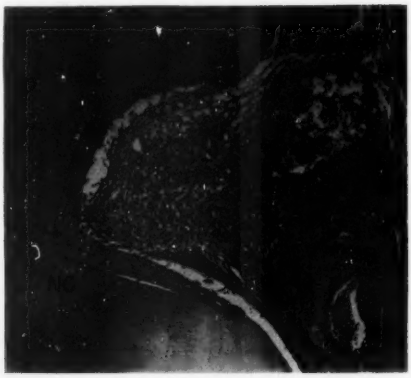

Fig. 1A. Inset of Figure 1 magnified. Thick layer of new cellular cementum (NC).

Biopsies were performed in the following manner. Two labial vertical incisions were made, one at each side of the healed surgical pocket. The two incisions were then joined about two millimeters apical to the estimated end of surgical separation. Modifying the technique of Workman,[24] the bony trabeculae included in the operative area were separated from the remainder of the alveolar process by following the lines of incision with a No. 557 cross-cut fissure bur, cutting into the root to insure complete isolation. To avoid trauma to the clinging tissue, the tooth was delivered labially with a straight elevator applied to the palatal surface of the root.

The extracted masses were fixed in Bouin's Solution, decalcified, imbedded in paraffin, and all were stained with hematoxylin and eosin except Case No. 5, in which Masson's tri-chrome was utilized.

RESULTS

A. Clinical—Chart I, a clinical résumé, reveals the subsequent results.

1. Apparent clinical reattachment occurred in all except case No. 6.

2. Cases No. 2, 3, and 13 showed more reattachment than surgical detachment.

3. The remaining cases (Nos. 1, 4, 5, 7,

Fig. 2. 42-day specimen, with cementum removed. Connective tissue fibers (CT) parallel to root. New cellular cementum (NC) especially prominent in nick (N).

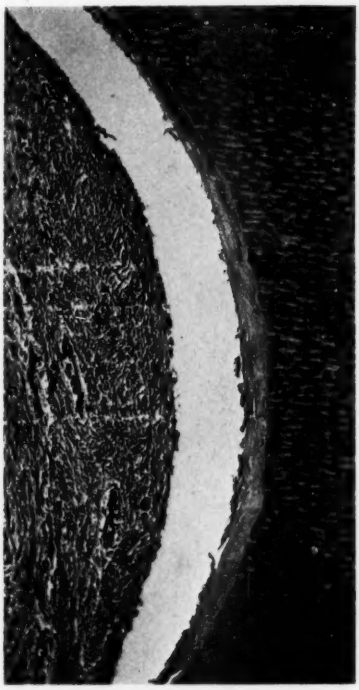

Fig. 2A. Inset of Figure 2 magnified. Thick layer of new cellular cementum (NC).

17, 18, 19) reattached up to an average of 1.2 millimeters less than the surgical detachment.

4. The seven cases (Nos. 1, 2, 3, 4, 5, 6, 19), where gingival marginal positions were noted, manifested two with no post-operative recession, four with recessions of one millimeter, and one with 0.5 millimeters.

5. The labial tissues of three cases (Nos. 1, 2, 3) were torn during extraction leaving five (Nos. 5, 7, 13, 17, 18) suitable for histological study.

B. Histological

Case No. 7 (Fig. 1), a 56-day specimen, is a simple surgical pocket without any further instrumentation other than the creation of the terminal nick (N). A prominent clear pink staining layer of cementoid (X) appears on the old cementum (C), thickening as it approaches N and becoming cellular cementum (NC) on the dentin (D) in the nick. Alveolar bone (AB) appears growing toward the concavity of the nick. The connective tissue fibers (CT) run parallel to the root, below and above the alveolar bone (AB). Artefacts (A) are noted in several places.

Fig. 1A is a higher magnification of the nick (N) on the dentin (D). Connective tissue fibers (CT) are noted parallel to the root surface near alveolar bone (AB).

Case No. 5 (Fig. 2) is a 42-day specimen (Masson tri-chrome stain) from which cementum was removed. The absence of an epithelial lining presumes the tissue to have been attached, although partly torn off during extraction and shrunken in processing (A). Light staining new cellular cementum (NC) is present on the dentin

Fig. 3. 71-day specimen, cementum remaining near epithelial attachment (EA) and at C. Connective tissue fibers (CT) parallel to root above and below alveolar bone (AB).

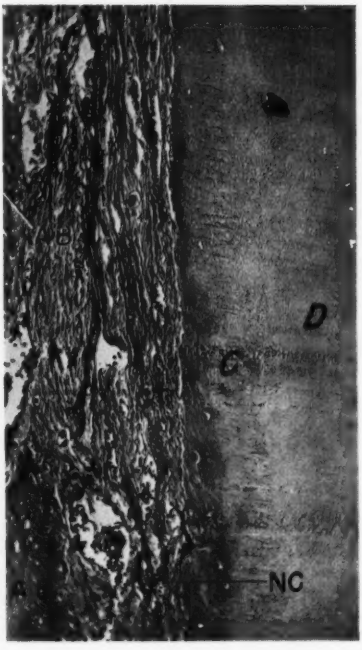

Fig. 3A. Inset of Figure 3 magnified. Connective tissue fibers (CT) parallel to root.

(D) in N. The connective tissue fibers (CT) run parallel to the root. Fibers not torn completely are noted at both ends of N.

Figure 2A, a higher magnification of N, shows cellular cementum (NC) deposited on the dentin (D) of the nick.

Case No. 17 (Fig. 3) is a 71-day specimen, with cementum ostensibly removed. Old cementum (C) remains apical to the epithelial attachment (EA) and immediately coronal to the nick (N). New Cellular cementum (NC) is present apical to the nick (N), deposited against old cementum (C). Connective tissue fibers (CT) run parallel to the root both above and below alveolar bone (AB). Shrinkage artefacts (A) are noted.

Fig. 3A is a higher magnification of the area of new cemental growth, apical to the nick. The parallel direction of the connective tissue (CT) may be plainly seen.

Case No. 13 (Fig. 4) is an 83-day specimen whose root was curetted to eliminate clinging periodontal fibers. Inspection shows, however, that cementum (C) remained only from the epithelial attachment (EA) to a point about halfway to the nick (N). Cementum was also removed beyond the nick (N) to N'. A thick layer of cel-

Fig. 4. 83-day specimen, cementum (C) remaining from epithelial attachment (EA) to point midway to nick (N). Connective tissue fibers (CT) parallel to root. Ned cellular cementum (NC) in nick (N).

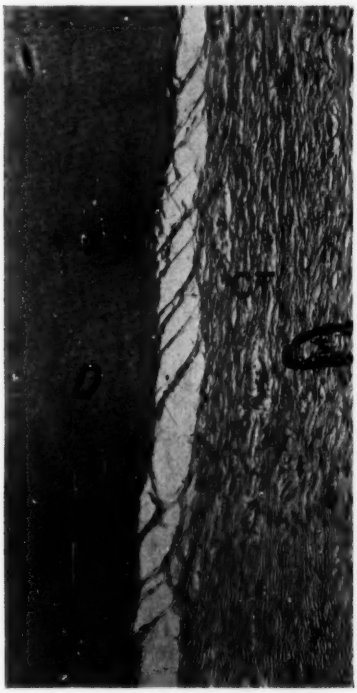

Fig. 4A. Inset of Figure 4 magnified. Deposition of acellular cementoid (X) on dentin (D).

lular cementum (NC) is deposited on dentin in the depth of N. Connective tissue fibers (CT) run parallel to the root.

Figure 4A is a higher magnification of N', showing deposition of pink, light staining cementoid (X).

Case No. 18 (Fig. 5) is a 106-day specimen in which the coronal half of the root was curetted and the apical half stripped of cementum with a bur. Examination of the section shows that old cementum (C) is present at the epithelial attachment (EA), is absent for a short distance below, then reappears as a thin layer, gradually thickening as the nick (N) is approached. Clear light pink-staining cementoid (X) is present on dentin (D) at the coronal portion of N. A gradually thickening layer of cementoid (X) also appears apical to N, on old cementum (C) becoming cellular cementum (NC) in the most apical portion. The direction of the connective tissue fibers vary, being parallel at CTP, and generally perpendicular to the root in the remaining and larger areas. The surface of the alveolar bone (AB) opposite N is lined with osteoblasts and has grown in toward the root defect.

Fig. 5A is a high power magnification of the area coronal to N, showing the generally mixed but transverse direction of

Fig. 5. 106-day specimen, cementum remaining coronal to epithelial attachment (EA) and coronal to nick (N). Connective tissue fibers definitely parallel to root only at CTP.

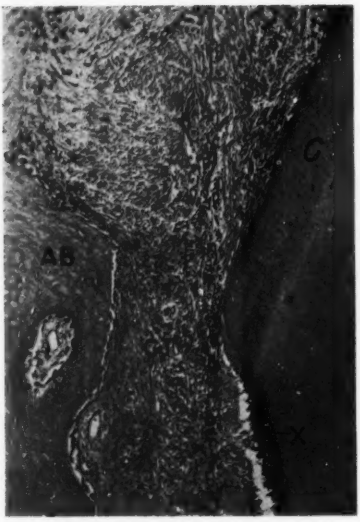

Fig. 5A. Inset of Figure 5 magnified. Connective tissue fibers (CT) more closely resemble normal functional orientation than in previous specimens.

connective tissue fibers (CT), and cementoid (X) deposited on old cementum (C) and dentin (D).

DISCUSSION

A. Clinical

The three cases where there was apparently more reattachment than detachment cannot be explained. In most cases there is a tendency for slight recession of the gingival margin and consequently slightly less attachment than detachment.

Case No. 6 was the only tooth showing no clinical reattachment. The tissue may have been loosened by trauma or inflammation. No biopsy was obtainable.

B. Histologic

Connective tissue healing took place in all surgical pockets against both dentin and cementum. This is in agreement with Linghorne and O'Connell's work on dogs[18] and contrary to Skillen and Lundquist's.[24] The first group of workers have explained this as possibly being the result of employing mandibular teeth (which did not reattach in their own experiments) due to the higher muscle attachments and the inclusion of hairs rubbed in by the animals' paws.

The selective reattachment to cementum alone noted by Stones[25] in monkeys was not true of humans. Experimental results cannot be transferred in toto from one species to another.

All sections showed connective tissue parallel to the root regardless of the relative height of the alveolar bone, or fiber contact with dentin, untouched cementum, curetted cementum, cementoid, or new cellular cementum, except at 106 days, the oldest specimen (Fig. 5). In this case, but for a small area (CTP) there is a mixed but generally transverse direction, with no correlation to the nature of the adjacent root surface.

Those fibers coronal to bone are part of the gingival groups described by Goldman,[14] some of which normally run parallel to the root. However, the fibers of the periodontal membrane traveling from bone to cementum are generally perpendicular to the root. This functional fiber orientation, under the conditions of this experiment, would seem to be dependent solely on time of healing.

Cementoid and cellular cementum are deposited on dentin and on curetted and non-curetted cementum. The thickest deposits and most numerous cellular elements were in the most apical regions, especially beyond the nicks, in the deepest dentinal nicks, and on uncuretted cementum. Similar observations have been noted by others.[18, 21]

The sections show that clinical intent may not correspond to histologic reality.

First of all, curettage may smooth cementum or remove it (Figs. 4, 5). This agrees with Riffle[23] and Thebaud.[27] Moreover, deliberate attempts at cemental removal may not be completely successful (Figs. 3, 5). Since cementum becomes thicker as the apex is approached[21] its unintentional removal is more likely to occur toward the cervical end of the root.

Secondly, the nicks intended to mark the end of surgical separation did not always reach this point, probably because of the width of the bur. This is shown by the removal of cementum beyond the nick in Figure 4. The similarly located marked cemental apposition in Figures 3 and 5 indicates a healing reaction that must have followed a recent wound. Furthermore, connective tissue fibers parallel to the root was observed apical to both nick and alveolar bone (Figures 1, 1a, 3, 3a). This is not the usual fiber direction and has been reported only in periodontal healing of surgical pockets[18] and pathological pockets.[12, 13]

The usual healing wound involves two vital surfaces producing bilateral bleeding and capillary growth, forming and supporting a clot that later becomes fibrous, then scar with partial or complete regeneration of the particular tissue. The wounds of surgical and treated pathological pockets present certain deviations from this situation. In both cases the soft tissue is vital, and provides unilateral bleeding and capillary growth. The root side, however, furnishes neither blood for clot nor capillaries for granulation tissue, and its vitality is a subject for discussion.

The physiology of dentin and cementum was discussed in a previous paper.[20] In brief, it was stated that because the cells in the deepest cemental layers die as new vital layers are added, it was evident that cementum receives its nutrition mainly from periodontal sources. When that was cut off by the formation of a pathological pocket the outer cementum also died. Therefore, one of the prerequisites of clinical procedures for reattachment of such pockets would logically seem to include cemental removal to expose vital dentin. Otherwise, epithelial downgrowth would interrupt any healing process. More recent work by Bass[2] has shown pathologic granules to appear in dentin and cementum opposite such a pocket, being most numerous near the dentino-cemental junction. This suggests removal of outer layers of dentin as well as cementum to reach vital tissue. The inadvertent exposure of vital dentin in curettage (Figures 4, 5) may have taken place in many of the reported successful clinical reattachments of pathological pockets, regardless of the avowed aim of the operator to merely smooth cementum.

SUMMARY

1. A technique was described for studying the healing of surgically detached periodontal tissues in man.

2. Connective tissue healing was observed in all teeth against uncuretted and curetted cementum, and dentin. The fibers were parallel to the long axis of the root in all cases except the oldest specimen (106 days).

3. Cellular cementum and cementoid were deposited on all types of root surfaces. The thicker and more cellular layers were generally noted in the more apical areas, the deepest dentinal nicks, and over uncuretted cementum.

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(Continued on page 264)

Temporary Splinting
An Adjunct in Periodontal Therapy

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TEMPORARY splinting devices have a useful place in periodontal therapy. Their use is indicated to obtain immobilization of the teeth quickly and easily to enhance healing following subgingival curettage, when reattachment is attempted, and to facilitate occlusal adjustments. In addition, these splints may have a diagnostic value, since teeth for which the prognosis is doubtful may be splinted simply and inexpensively for from several months to a year, after which an evaluation may be made as to the effectiveness of therapy. If the treatment is successful the teeth may be splinted permanently by a fixed appliance.

The anterior teeth may be splinted adequately by the use of stainless steel wire, the technique for which was reported by Hirschfeld.[1] The posterior teeth, however, cannot be splinted by this means. The cast continuous clasp device, either with retentive clasps or cemented without clasps, has been employed as a posterior temporary splint.[2, 3] This method has one serious shortcoming; it does not stabilize the teeth against axial or apical displacement. Where the splint is not cemented, some resistance to lateral displacement is gained, but not as effectively as can be accomplished by a fixed splint. Where the cast continuous clasp is cemented to the teeth more rigidity is obtained, but eventually, due to the axial forces exerted during function, the cement seal may be broken and the teeth pushed out of the splint. As a general rule, teeth which require splinting can be depressed in their sockets to some extent. It was felt, therefore, that this type of splinting should be modified to resist displacement apically as well as laterally, thereby making it as effective as a fixed appliance.

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DESCRIPTION OF SPLINT

Such a splint was devised by the author and found to be successful (Fig. 1). It is designed in such a way that the casting is both above and below the heights of contour of the teeth, thus stabilizing them against depression apically. To permit the splint's placement below the height of contour it is open at the anterior end. This allows the operator to spring it open sufficiently to seat the appliance, after which the anterior end is held securely closed by a stainless steel wire threaded through holes which are made in the appliance before it is placed (Fig. 2). The inner surfaces of the splint are coated with a slow-setting cement or periodontal pack prior to its placement and wiring to hold it in position securely. Placed in this way, such a device creates complete rigidity. It may be necessary to repeat the cementation after three or four months; if so, it is a simple matter to untwist the wires and remove the splint in order to do this.

Fig. 1. Cast continuous splint designed to immobilize the teeth completely. Casting is both above and below the heights of contour, thus preventing lateral movement and depression of the teeth in the alveoli. Note arrow which indicates the point at which the wires are twisted to close the anterior end of the splint.

Fig. 2. The same splint as in Fig. 1 on the model, showing the wire threaded through the holes made near the open end. These will be twisted after the splint has been sprung open and cemented into position.

A variation was tried wherein a hinge was made posteriorly to facilitate opening and closing the splint when placed over the teeth (Fig. 3). It was found to offer no advantage, complicated the construction, created more bulk, and was therefore discarded in favor of the splint described above. A modification of the cast splint can be made and used in a diagnostic capacity (Fig. 4). In this case some doubt existed as to whether the results of therapy would warrant fixed prosthesis, therefore the replacements for the missing anterior teeth were incorporated in the splint and the equivalent of a fixed bridge was created. This is to be left in position until sufficient time has elapsed to evaluate the periodontal therapy and splinting.

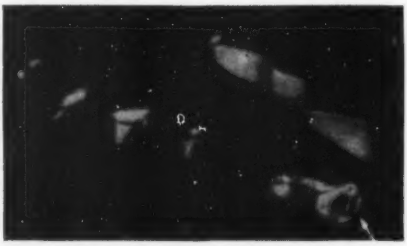

Fig. 3. Note arrow showing the hinge built into the splint to facilitate opening and closing. This had several disadvantages, and was discarded in favor of splint in Fig. 1.

ORTHODONTIC BANDS AS SPLINTS

A splint consisting of orthodontic bands soldered together will provide as much rigidity as the cast metal splint described above, but it has three disadvantages. First, if the teeth are in contact, they must be separated sufficiently to place two thicknesses of band material between them; second, the splint is more difficult to remove and replace if such a necessity arises; third, if the teeth are not to be splinted permanently after the removal of the bands, the teeth must be moved into contact again, and such movement in itself creates mobility.

Fig. 4. A modification used for diagnostic purpose. The outcome of the case was doubtful, and in order to obviate a fixed splint this device was placed as a provisional splint. It was cemented on and wired securely through the holes provided distally on both sides. The anterior teeth were also replaced with this appliance. (Courtesy of Dr. Robert L. Reeves)

SPLINT FOR REATTACHMENT ATTEMPTS

A temporary splinting device analogous to the cast metal splint described above can be employed to serve a dual purpose when attempts at reattachment are made in the treatment of the infrabony pocket, as reported by Goldman.[4] It will serve to protect the pack by keeping it firmly in

Fig. 5. Acrylic splint used when attempting to obtain reattachment in the treatment of the infrabony pocket. The same principle of an open end splint is used as in the cast continuous splint. This not only stabilizes the tooth to be treated, but protects the pack from being dislodged. It may be left in position undisturbed for 2 weeks.

position and preventing its displacement or movement, and at the same time it will stabilize the teeth. An acrylic splint is made extending around the distal of the last molar and anteriorly sufficiently to include the tooth to be treated (Figs. 5 and 6). It is left open anteriorly to permit springing the device sufficiently to place it, and then wired to close the anterior firmly. Relief is made on the inner surfaces to provide room for the periodontal pack; however, the peripheries are firmly in contact with the soft tissue and the teeth, to seal off the underlying pack and hold the teeth firmly in position.

This splint can also be made with self-curing acrylic. This method offers not only the advantage of conserving time, but the space for the periodontal pack can be easily provided during the construction. A model is made and an outline of the splint is drawn on it. Moldine is placed around the teeth and over the tissue area in the same position and thickness as the operator anticipates the periodontal pack to be. A separating medium is painted over the entire outlined area. The acrylic powder is poured on the model to cover a small section, and enough liquid to saturate the powder is added. More powder is poured on the cast and moldine, and liquid added. Thus with repeated applications of powder and liquid the entire outlined area is covered. The powder is best applied from a small container with a small spout or hole, so that its application can be easily controlled. The liquid is applied with a medicine dropper. After the acrylic has hardened it is pried from the model, trimmed, polished, and holes are drilled at the open end to accommodate the wire. Thus the same type of splint is constructed as shown in Fig. 5, except that the half round clasp wire around the distal is replaced by acrylic. There will be no need to grind out the inside of this splint to make room for the periodontal pack, since the moldine has created the necessary relief.

Since it is believed that immobilizing the tooth and protecting the blood clot during reattachment therapy is of the utmost importance, such a splint is a distinct aid in this phase of treatment.

Fig. 6. The acrylic splint in position. The tooth treated here was the first bicuspid. The infrabony curettage was done, foil placed over the area, pack placed over the foil, and the splint positioned and wired into place. It is felt that this offers the best possible protection to the blood clot, thus increasing the chance of success in reattachment. To prevent irritation of the lip, the twisted ends of the wire are countersunk into a groove cut in the acrylic.

INDICATIONS FOR USE

1. Immobilization of posterior teeth to
   A. Promote healing
   B. Facilitate occlusal adjustment

2. To provide splinting for diagnostic purposes.

3. To protect the pack and stabilize the teeth in reattachment attempts.

ADVANTAGES OF THESE TEMPORARY SPLINTS

1. The cast splint provides complete rigidity and prevents lateral and apical movement.

2. Ease and rapidity of construction.
3. Can be constructed economically.

SUMMARY

Temporary splinting is a valuable auxiliary procedure to employ concomitant with or following periodontal treatment. The splints described above have been found to be helpful in various phases of periodontal therapy.

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The Dentin: Its Physical Characteristics During Curettage

by A. B. RIFFLE, D.D.S., *Rochester, N. Y.*

THIS study demonstrates a previously unrecognized entity in the gross pathology of the human tooth; not a rare, or even an occasional condition, but rather, one so common as to make its complete absence the rarity.

Previous studies have shown that the enamel surface in the cervical area of most teeth is definitely "crusty" and readily removed during the repeated passage of a sharp curetting instrument over its surface, and that this is usually so deep at the cemento-enamel junction that there is a measurable loss of the enamel in its entirety;[1] that the same repeated passage of a sharp instrument over the cemento-enamel junction resulted in complete loss of the cementum in this area;[2] and that the same procedure over the cementum of the pathologic crevice area resulted in complete loss of the cementum.[3] In each of these studies it was mentioned that not only was the enamel lost in part, and the cementum in its entirety, but also that definite areas of dentin were removed as well.

Interest in these studies developed during the author's early attempts to curet and plane the root surface of the pathologic crevice area in conformity with the usual teachings. We had been instructed to remove all calculus, organic debris, etc., from the tooth surface in the crevice area, and this proved to be easy (including calculus). However, instructions to plane the *surface* of the cementum to a depth just sufficient to remove the necrotic remains of the periodontal fibers proved to be impossible.[2,3] Try as he would, he would find his curetting instrument "catching" on a hard ledge of enamel and would know that he had gone deeper than the rules allowed.

Hoping to improve his finger skill, he followed Stillman and McCall's recommendation[4] that we practice on extracted teeth and found that curetting the root surface to a hard, smooth, "velvety" surface,[5] like "polished ivory"[6] resulted in a definite circumferential grooving in most teeth—a groove of a definite characteristic pattern, identical in shape to the classical wedge-shape abrasions of Miller,[7] but occurring

on all surfaces of the tooth. Had these grooves been of the same depth all around every tooth, or even most of the teeth, these findings might be discounted, but there seemed so many variations in incidence, in depth, in degree of softness during curettage, etc., that a formal study seemed justified.

METHOD OF STUDY

A plan was evolved in which 500 extracted teeth from the mouths of 312 patients were painstakingly examined and curetted. Though these teeth were taken just as they presented in the routine day of 3 oral surgeons, there seems to be a sufficient number of different kinds of teeth, of different age groups, of different reasons for extraction, and of different degrees of periodontal involvement, that average findings should represent definite trends. The systematic grouping of these teeth has been previously published.[1]

A printed form was prepared on which one could record the name of the tooth, the age and sex of the patient, the reason for extraction, the condition of the tooth, calculus deposits, and the character of the tooth surface before, during, and after curettage. The form also included measurements to .5 mm of the greatest diameter bucco-lingually and mesio-distally, both at the cemento-enamel junction and at the bottom of the pathologic crevice, and also the distance from the cemento-enamel junction to the bottom of the pathologic crevice (Crevice Depth) and to the apex on each surface.

Two x-rays were taken of each tooth—mesio-distally and bucco-lingually. Four photographs were also taken, one of each surface, and, after curettage, four more in the exact position as before curettage. This served as a check on measurements and provided visual confirmation of the results.

Each tooth was then carefully curetted in the area of the pathologic crevice, repeatedly passing the *sharp* blade over the tooth surface with moderate pressure until the author obtained the hard, shiny "feel" interpreted as healthy tooth. When this point is reached, there is no longer a "gritty" feel to the instrument, nor an audible "crunching," nor a visible "powdering" of the surface, but rather a smooth, "glassy" feel and sound as if one were passing the instrument over a glass table top. Standard curetting instruments were used at first, but this soon proved too arduous, and the study was finished by scraping with a large Kingsley vulcanite scraper. The results are the same.

During the process of curettage, observations were recorded as to the character of the surface, such as visible changes in color and general appearance, whether soft or hard, whether the material removed seemed to be a powder or more like shavings, and whether it was difficult to remove or readily scraped away.

The first few strokes of the sharp instrument completely removed the cementum in most cases, whether a pathologic crevice was present or not[2,3] and at the same time on most teeth a definite area of the enamel surface was removed in the form of a fine powder, a measurable amount of the enamel at the cemento-enamel junction being lost in its entirety.[1]

GENERAL OBSERVATIONS

This left a tooth on which the dentin was exposed from the bottom of the pathologic crevice, as represented by the clinging attached fibers of the periodontal ligament, to the New Enamel Line (after the measurable loss of enamel described before). At this stage, the appearance of the tooth is not markedly changed except for an accentuation of the cemento-enamel junction, and on many young teeth, as well as on some from the mouths of older patients, this is the end of curettage, for one immediately encounters the hard "glassy" feel the author strives to attain (Figure 1).

On most, however, as the scraping continues on the dentinal surface, there is no distinguishable difference in the "feel" of

Fig. 1. Upper pictures, Before Curettage from buccal, lingual, mesial and distal. Lower pictures, After Curettage from buccal, lingual, mesial and distal.

the tooth surface, or in the character of the shavings removed. In other words, one cannot distinguish between curetting cementum and curetting dentin, as Box[8] recognized long ago.

On teeth which are definitely involved, one notices a characteristic pattern forming. At the bottom of the pathologic crevice, the instrument "bites" for only a few strokes, and then slides over this area with velvety ease, just as it did at the cemento-enamel junction in most young teeth. In the area adjacent to the enamel, however, the instrument continues to cut and shave, penetrating deeper and deeper at the original location of the cemento-enamel junction. On teeth with little involvement, the tendency is for the formation of a shallow V-shaped "ditch," circumferentially around the tooth, especially on the buccal surfaces (Figure 5); on those more seriously involved, this circumferential groove may be very deep (Figure 7).

The most serious involvement is found in teeth with visible caries at the cemento-enamel junction. In these, the groove formed by the removal of the carious area and the softened dentin beneath, frequently results in pulp exposure and even in fracture before the hard, smooth feeling is at-

tained. The same characteristic—frequent pulp exposure and fracture—is also found in teeth long exposed to the ravages of periodontal diseases (Figure 7), even when no surface changes are evident, and also in "periodontosis" cases in the comparatively young (Figure 2).

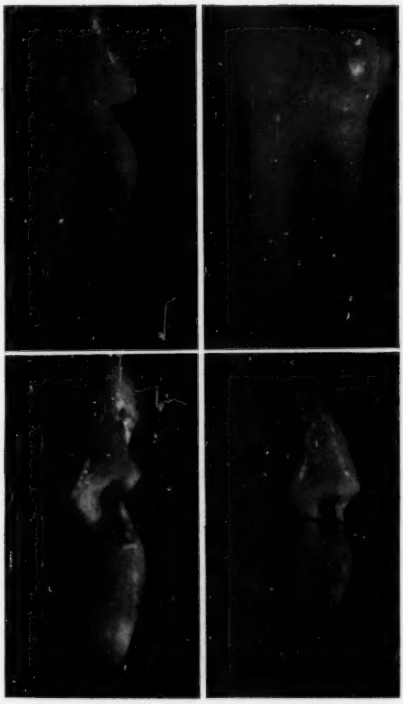

Fig. 2. Upper left, Deep Penetration at 15 years. Upper right, Slight Penetration at 55 years. Lower left and right, Pulp Exposures at 25 years.

DEFINITIONS

Definition of the characteristics of the lesion will aid in understanding the rest of the study.

The *characteristic pattern* of the lesion (Figures 3, 5, 6, and 7) is a circumferential groove, V-shape in character, the deepest part of which takes the form of a line at approximately the original location of the cemento-enamel junction; the apical wall of which is a more or less gradual slope from the original circumference of the root at the bottom of the pathologic crevice to this line of deepest penetration; and the incisal/occlusal wall of which is a surface at varying degrees of angulation from the new enamel line to the line of deepest penetration.

The *line of deepest penetration* is the circumferential line formed at the deepest portion of the penetration (the apex of the V). (See Figures 3, 5, 6, and 7).

On some teeth the depth of the penetration into the dentin is about equal completely around the tooth but on most, one area in the groove is markedly deeper and wider than on the other surfaces. This is called the *"area of deepest penetration."* (See Figures 3, 6, and 8, lower right).

On some teeth with an Area of Deepest Penetration, there is another area which is not as deep as the above, but still deeper than the depth of the groove on other surfaces and possessing all of the other characteristics of the above. This is called *"the area of lesser penetration"* (not illustrated).

THE GENERAL CHARACTERISTICS OF THE LESION

Watching the character of the tooth during the planing, one will notice that if any considerable degree of softened dentin is present, the area which will become the Area of Deepest Penetration will feel definitely softer to the curetting instrument than do the adjoining surfaces, and that the loss of dentin is greater on both the incisal-occlusal and apical walls of the V. In the floor of this area, as the scraping continues, there is usually a "cloudy," whitish or yellowish appearance, distinctly different from the translucent dentin around it. In the center of this "cloudy" area there frequently appears a small spot, yellowish or brownish in color, reminding the observer of the brown or yellow spots described by Gottlieb in advance of beginning caries. This spot disappears with con-

tinued curettage, as does the "cloudy" area, if hard smoothness is attained. If hard smoothness cannot be obtained, pulp exposure occurs in the center of the "cloudy" area, directly under the brown or yellow spot. These variations from the normal are almost always found under areas of cervical caries, but are also prevalent on most seriously involved teeth in the absence of any visible surface changes. (Figure 7).

On other teeth, as one observes the character of the tooth during scraping, instead of the cloudy appearance noted above, one encounters an area which is definitely more like "horn" than the surrounding dentin. Still translucent, it is of a different degree of translucency, more like the eburnated appearance of the walls of an old wedge shape abrasion. This is interpreted to be sclerotic dentin—an area in which nature's defenses have been more successful than in the cloudy areas above. If deep enough, one may also penetrate to secondary dentin.

The same characteristics will also be found in a lesser degree in the areas of lesser penetration defined above, and on some teeth from mouths in which periodontal involvement is exceptionally severe they will be found on all surfaces of the tooth, penetrating to the pulp and resulting in actual fracture of the tooth as curettage continues (Figure 2).

Still another characteristic is a definite tendency for the softening of the dentin to undermine the enamel at the cemento-enamel junction, and the removal of this softened dentin accounts in part at least for the loss of enamel at the cemento-enamel junction described in our studies of the cervical enamel on this same series of extracted teeth.[1] It also accounts for the sharp ledge of enamel which develops at the chair when curettage has "gone too deep."

The depth of these penetrations varies not only on different surfaces of the same tooth, but from tooth to tooth in the same mouth, with age, and apparently with the intensity of the attacking process and the

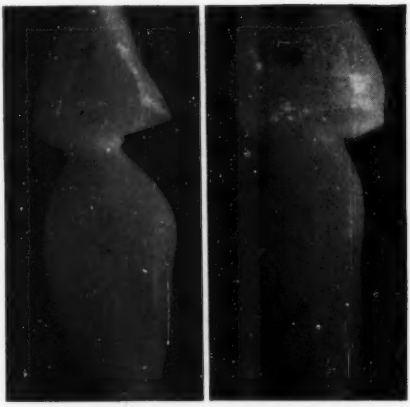

Fig. 3. The Characteristic Pattern.

individual's own resistance to this process. Thus, *Figure 2* shows a rather deep penetration at fifteen years, two teeth with exposure at twenty-five years, and a tooth practically free from involvement at fifty-five.

For purposes of study, however, most teeth follow a general pattern, whether classified into Age Groups, or according to the Depth of the Pathologic Crevice. *Figure 1*, for example, is typical of the findings in youth, though this particular tooth is from the mouth of a white female, forty-seven years of age and was extracted because of periodontal involvement. *Figure 4* presents three teeth from children, extracted because of caries. None of these teeth show much more than a slight exaggeration of the cemento-enamel junction after curettage and probably closely approximate the normal.

Figure 5 represents the early stages of the deterioration, typical of the sixteen to twenty-five year age grouping, though this particular tooth is from the mouth of a white female, ten years of age, and was extracted because of deep caries. Here is a definite *line of deepest penetration*, 1 mm loss of enamel at the cemento-enamel junction, a penetration of .5 mm completely

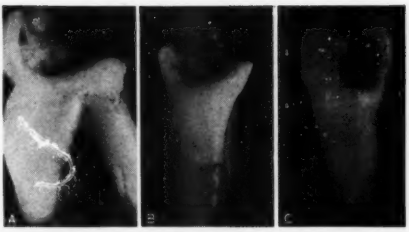

Fig. 4. Large cavities adjacent to the cemento-enamel junction.

around the tooth, and no area of deepest penetration.

Figure 6 is found quite frequently after 30 years of age, this tooth being from the mouth of a white female, fifty-two years of age, which was extracted for periodontal reasons. On this tooth one finds the usual definite *line of deepest penetration*, a variation in the amount of enamel lost at the cemento-enamel junction (a slight loss on the buccal; a 2 mm loss on the lingual; a 1 mm loss on both mesial and distal), and *an area of deepest penetration* on the mesio-lingual surface, correlated with the area in which the amount of enamel loss was the greatest.

Figure 7 is representative of many teeth in late life, this tooth being from the mouth of a white female, fifty years of age. The tooth was extracted for periodontal reasons. Here is found a marked enamel loss on all surfaces, the *line of deepest penetration* obscured by the line of fracture, and an *area of deepest penetration* on the mesio-buccal, so deep that exposure of the pulp and fracture of the root occurred before the hard smooth surface was attained.

RELATION TO WEDGE-SHAPE ABRASIONS

At a glance these characteristic patterns are the same as the wedge-shape abrasions so commonly seen in practice, except that they are found on all surfaces of the tooth, and not just on the buccal where most wedge-shape abrasions are found clinically. Miller[7] taught that the pattern was caused by the friction of the brush and an accompanying abrasive or chemical reaction on a *normal* tooth. As a result of this study, it seems more likely that the pattern is formed on a *previously deteriorated area* on the tooth. For example, a specially built machine was demonstrated at the American Dental Association meeting in Chicago in 1949—a machine in which extracted teeth could be brushed at predetermined pressures and which would compute the number of strokes automatically. The teeth being brushed were not wearing away alike; some were grooving deeply—some barely perceptibly, though they were side by side with the same number of strokes from the same type of brush at the same pressure.

RADIOGRAPHIC CONFIRMATION

There is other evidence of the existence of this pathology. *Figure 8* shows that these deteriorations, usually undermining the enamel, are visible on the x-ray negative. Called "Normal Cervical Radiolucency" by Raper,[9] a study of the postextraction x-rays (both bucco-lingually and mesio-distally) compared with the characteristic pattern formed on that tooth during curettage shows a definite correlation between the two.

On the x-ray negative the lesion varies from a small, faintly darker area at the cemento-enamel junction (Figure 8, A and B) to a denite delineation of the area in the more severe (Figure 8, C and D). Visible over 90% of the time, the x-ray did not always show the same degree of penetration as the post-curettage results.

Of course, in the mouth, exposures through the mesio-distal diameter, cannot be made through the buccal and lingual areas—the areas of earliest, most frequent, and deepest penetrations. From the findings in this study, however, one can be sure that if penetrations are visible on either the mesial or distal surfaces, or both, greater penetrations will be found on either/or the buccal and lingual surfaces.

Fig. 5. Upper pictures, Before Curettage from buccal to distal. Lower pictures, After Curettage from buccal to distal.

OTHER EVIDENCES OF THE PRESENCE OF THE LESION

Recently, Wainwright[10] has demonstrated the permeability of tooth structure to various radioactive solutions, the radioautographs showing "marked penetration of exposed cervical dentin to the pulp chamber," a decidedly greater penetration in the pathologic crevice area than in the rest of the tooth. This is the first time we have seen the characteristics of this change in the dentin demonstrated by any other means than by the planing technic herein described. Still more recently he has shown the same pattern of penetration in teeth extracted from patients who have ingested radioactive iodine solutions for the treatment of malignancies of the thyroid.[11]

Bass[12] probably has described part of the histologic characteristics when he recently noted a "Previously Undescribed Demonstrable Pathologic Condition in Exposed Cementum and the Underlying Dentin." This consists of a peculiar granular condition found only in and under cementum exposed to a pathologic crevice and never in wedge-shape abrasions, and which can only be demonstrated by a different technic of preparing the tooth for histologic examination.

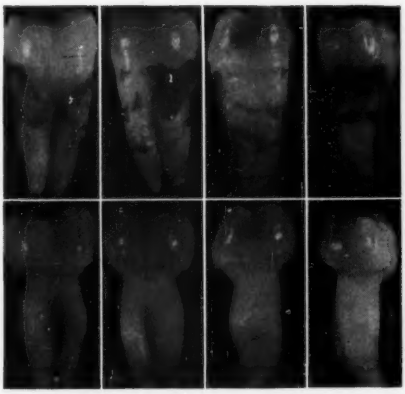

Fig. 6. Upper pictures, Before curettage from buccal to distal. Lower pictures, After curettage from buccal to distal.

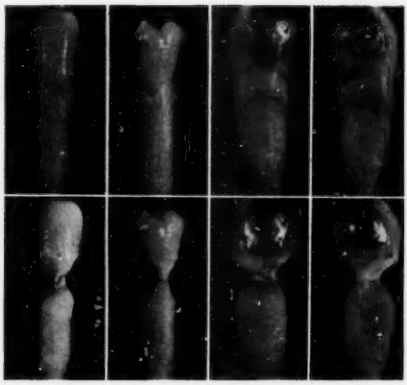

Fig. 7. Upper pictures, Before Curettage, from buccal to distal. Lower pictures, After Curettage, from buccal to distal.

The author has never been able to see a simulation of the characteristic lesion on either decalcified or ground sections of teeth prepared by the usual methods, even though several years ago Dr. H. K. Box spent considerable time going over his large collection of histologic sections without being able to demonstrate its existence.

Cahn,[13] in 1926 noticed abnormally large channels running from the surface of the root, directly to the pulp in "pyorrhetic" teeth, and believed them to be the result of either bacterial invasion or the action of bacterial end-products. Probably these are also histologic evidence of the softenings just described.

There is considerable clinical evidence of the presence of these cervical changes. Firstly, as mentioned before, there is the common wedge-shape abrasion, which the author now believes to be the abrasive loss of a previously deteriorated area. These are not necessarily the result of abrasion, however, for the experienced periodontist frequently feels these wedge-shape areas with his curetting instrument at the beginning of subgingival curettage, completely covered by the free gingivae and consequently not exposed to toothbrush abrasion. Neither is there a history of previous curetting or scaling.

Even more common is the ease with which the dental bur cuts tooth structure in the preparation of cervical cavities, the bur rapidly making its way in all directions laterally from the actual carious area in comparison to the difficulty of cutting healthy dentin in other areas of the tooth.

Also, there is the toothache in a tooth without either dental caries or a filling of any kind. Adequate subgingival curettage relieves the pain almost immediately, especially if carried out to the extent of removing the deteriorated areas just described. In other words, the pulpal irritation was apparently caused by some external factor with some means of dissemination to the pulp or the removal of this factor would not have relieved the pain. This same factor —long standing or acute pulpal irritation from periodontal involvements—probably also accounts for the occasional apical rarefactions discovered on routine x-ray examination on a tooth without either caries, filling, or history of trauma to account for the pulp death.

DISCUSSION

Little wonder that this gross pathology has escaped previous detection. As one examines the pathologic cervice area on an extracted tooth, it ordinarily presents a

hard-appearing, shiny surface, no different from the rest of the root except for the clinging shreds of the fibers of the periodontal ligament on the latter. It may be partly covered with calculus, but when the calculus is removed there is the same hard, shiny-looking surface beneath it. There may be slight roughnesses with moderate magnification, but we have been taught that the *surface* of the cementum is injured and roughened during long exposure to the contents of the pathologic crevice, so this excites no suspicion. And if caries is present, one thinks of it as being unrelated to the periodontal problem and dismisses it.

Under the microscope the lesion has not been seen for the usual methods of preparing teeth for study do not show it. Roughness of the surface of the cementum in the pathologic crevice and actual loss of the cementum are frequently seen histologically, but seem to have excited little interest.

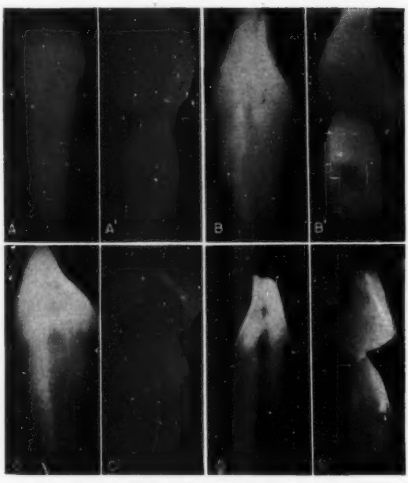

Fig. 5. Curetted teeth (A^1, B^1, C^1, D^1) and corresponding radiographs (A, B, C, D).

Then, we have been cautioned so many times not to curet through the cementum —only to remove the "outer third"—impossible as we have demonstrated this to be. Consequently, there has been no interest in curetting further, though all periodontists have been doing it inadvertently, by repeated curettage, if not at the first sitting,[3] unable to tell by the feel of the instrument whether they were cutting calculus, cementum, or dentin.

In other words, the tendency is to look at the tooth and declare it normal because it "looked normal," only recognizing as abnormal, visible lesions such as decalcifications, caries, and abrasions. During curettage of the root surface the "crunchy," "gritty" sound and feel is interpreted as always being calculus because it sounds and feels like calculus. The smooth, "velvety" feel attained in successful curettage has been thought to be the surface of the cementum, when in reality the cementum has been removed and the dentin penetrated for various depths. The author thought he was doing wrong when he felt "grooves" developing at the cemento-enamel junction during curettage, but now believes it is mandatory to develop these "grooves" if it proves impossible to obtain the hard smooth "feeling" to sharp curetting instrument without doing so.

CLINICAL SIGNIFICANCE OF THE LESION

These softened areas seem to the author to be analogous to dead or dying bone, and as such could only be a foreign body, exciting the body's usual reaction to a foreign body—chronic inflammation in the adjoining tissues. In studies of deteriorated enamel,[1] the cemento-enamel junction,[2] and the cementum,[3] in the pathologic crevice area, the author concluded in each case that their removal is obligatory, and now concludes that the removal of the deteriorated dentin is equally imperative. A foreign body can only be a chronic irritant. It is good surgery to remove it. The author has been doing so for over ten years, and becomes more enthusiastic with each passing year. Others have been doing it unknowingly, thinking they were doing something wrong if they realized they were doing it at all. A close study of successfully treated cases, carefully compared with those not as successful will soon convince the skeptical.

The most certain cure for periodontal disorders is to remove the tooth. The next most certain is to completely remove the part of the tooth which is the site of the irritation that causes the chronic inflammations that periodontal disorders are.

It is customary to describe the periodontal pocket as consisting of two walls; one, the tooth surface—the other, the soft tissue surface. Then it is usually stated that obviously the tooth surface is a passive factor. Volumes have been written on the study of the soft tissues and bone under the microscope, and they all agree; nothing is found but the various stages of inflammation and the expected sequel of events in chronic inflammatory processes. Then, with the greatest of inconsistency, treatment is discussed. The tooth surface is planed to smoothness. Removal of the soft tissues alone will fail; adequate curettage of the tooth surface without removing the soft tissues will succeed. When one removes the *source* of the irritation (the deteriorated tooth surface) the soft tissues and the surrounding bone return to normal; when one removes the *result* of the irritation (the chronically inflamed soft tissues), the gingivae grow back to the same size and location that they were before their removal, unless one is able to keep the surface of the tooth absolutely clean for long periods of time, a situation which most patients find impossible until these deteriorated surfaces are removed.

Thus, the tooth surface of the pocket is not a passive factor. It is the active factor.

CONCLUSIONS

There is a previously undescribed softening in the cervical dentin of human teeth, which when planed away with a sharp instrument, as in periodontal root curettage, presents a characteristic pattern—the same pattern as wedge-shape abrasions, but on all surfaces of the tooth in most cases.

Five hundred extracted teeth were curetted in the pathologic crevice area, the general observations and results are recorded and discussed, and the characteristic pattern of the lesion is described in detail.

Slight, or nonexistent, in children's permanent teeth, the depth of the softened areas increases progressively with the years and with the increasing depth of the pathologic crevice.

The clinical significance of the lesion is discussed and the recommendation made that the deliberate removal of these softened areas is mandatory for truly successful periodontal treatment.

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The Etiology of Periodontal Disease

by PAUL E. BOYLE, D.M.D.,* *Philadelphia, Pennsylvania*

ETIOLOGY literally means a discourse on causes, or, the study of causes. When causes are mentioned, we immediately think of effects, indeed, the study of causes usually comes after the study of effects or results. Between cause and result there is a time period which may be short or long and during this period various processes occur. The cause or causes may be thought of as trigger mechanisms which set in motion complicated reactions. In dealing with disease we are concerned largely with causes which produce various degrees of injury to the host. The reactions to injury produce signs and symptoms of disordered function. These reactions to injury appear to have as their purpose restoration of the normal state of health. A disease entity is described by Forbus[1] as "the sum total of the numerous expressions of one or more pathologic processes." Pathologic processes are grouped under the term "pathogenesis." They are responsible for the signs and symptoms of disease. We may summarize these relations in the following way:

Etiology	*Pathogenesis*	*Disease*
Injury producing agents. (Numerous)	Processes or responses to injury. (Few)	Results of responses to injury.

Etiologic factors concerned with disease are not only numerous, but they are often interrelated. For example, mechanical trauma may be associated with bacterial infection, or infection by one microorganism may pave the way for invasion by another, as in streptococcus infection following influenza.

Forbus has grouped the reactions of the body to injury into three categories; submissive, reactive and adaptive. The submissive reactions are essentially passive, the damage done to the tissues by an injurious agent. This may be minimal or severe. It may be momentary, as with a single blow, or repeated. It may be due to living or nonliving agents. Reactive responses include the processes of inflammation and repair. Adaptive responses are all the compensatory mechanisms which are called into play to enable life to continue in the presence of injurious agents.

From a somewhat different point of view, diseases may be classified in two great groups.[2] In group one, identification of the etiologic agent is of paramount importance in recognition of the specific disease. Infections, traumas, poisonings and allergic manifestations are examples of diseases resulting from the action of diverse etiologic agents.

Group two diseases are recognized and classified on the basis of structural changes in the tissues or alterations of function. Degenerative, metabolic, neurological and neoplastic diseases as well as abnormalities of development are included in this group. Here the etiologic agents are not known or are so complex as to be of little value for identification and classification.

Etiology in group one carries a suggestion of simplicity, find *the* cause, eliminate or counteract it, and one has *the* treatment and perhaps even *the* method of prevention. The triumphs of bacteriology have given credence to this oversimplification. The diphtheria bacillus growing on a body surface causes the disease diphtheria through the production of local and circulating toxins. When these toxins are neutralized by the use of anti-toxins, the disease loses its terrors. Susceptible individuals are identified (Schick Test), immunity is established by appropriate treatment, and the disease practically disappears from the morbidity and mortality tables.

Read before the Academy of Periodontology, September 4, 1952.

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The disease typhoid fever, to cite another example, is caused by Salmonella typhosa through the production of endotoxins. Immunity to the disease is gained through the use of vaccines, transmission is prevented by sanitation, and again a once powerful killer becomes a clinical rarity. Even with these diseases, however, the reasons for relative immunity of certain individuals and the great natural resistance of most animals other than man, indicate that there are complex factors involved in spite of a superficial appearance of simplicity.

Hope of discovery of this cause and effect type of etiology for periodontal disease flared briefly with the demonstration of *Endamoeba gingivalis* in the oral cavity and in pyorrhea pockets. The use of emetine as a more or less specific amebacide, however, proved disappointing and it is now recognized that these amebae are nonpathogenic.[3]

Other "causative" micro-organisms show clearly a more complicated situation. Staphylococci, for example, are constantly present on the skin. Once they gain entrance to the deeper tissues they are capable of producing abscesses. Yet, in most individuals, they are kept from causing injury by the acid secretions, by the outer scale cells of the epidermis and by other local factors, which promote immunity. Even in particularly susceptible individuals, furuncles or abscesses usually occur only in localized areas and their appearance often seems to be closely related to other factors in addition to the presence of the micro-organisms. Thus pimples may appear where a collar rubs the back of the neck. They usually occur about hair shafts. The palms of the hands and other locations where hair is absent, practically never develop pimples. Pimples often first develop at puberty, a time when the hair and related skin appendages are developing rapidly under the influence of the endocrine glands. Diabetic patients are particularly susceptible to the development of abscesses. Patients with nutritional deficiencies are also liable to furunculosis.

Staphylococci together with other potential pathogens are also constantly present on the oral mucosa, yet in most instances they remain on the surface and do not cause disease. Conditions which predispose to the invasion of the oral tissues by pyogenic oral micro-organisms and the development of periodontal abscesses are not well understood.

Enzymes of the hyaluronidase or collagenase groups may play a role in effecting invasion through the normally intact epithelial barrier. The efficiency of phagocytosis of bacteria by neutrophils is regulated by opsonins and other immune bodies. In health phagocytes constantly ingest and destroy the small numbers of pathogenic micro-organisms which gain entrance to the tissues. The phagocytic cells have often been compared to an efficient police force in a large city, which is constantly detecting and arresting criminals so that the peaceful citizen is undisturbed. An abscess may be compared to a situation where the criminals have been allowed to become so well established that a pitched battle is necessary to bring them under control.

ANATOMICAL FACTORS IMPORTANT

Anatomical factors are also important in the initiation and progress of disease, much as slums and poor lighting conditions predispose to crime. The gingival crevice like the junction of the skin and hair shaft is a point of lessened resistance to the entrance of micro-organisms.

We must also consider nutritional and endocrinological states since these govern to an important degree the ability of the tissues to resist infection and to overcome infection once established. The analogy to social and economic conditions in relation to criminality appears apt. Criminals are much more numerous when economic and social maladjustments prevail. The importance of nutritional and endocrinological disturbances as predisposing factors to the development of periodontal diseases has been noted by many periodontists.

The control of staphylococcal infections of the skin and of the somewhat similar infections about the teeth has proved to be a difficult clinical problem. Recognition of potentially pathogenic micro-organisms is only one step, since such organisms are universally present on skin and mucous membrane. Their numbers may be reduced temporarily by soap and water or other cleansing, mildly bactericidal, agents and by judicious scrubbing. This is one of the methods by which the periodontist attempts to prevent or control periodontal disease, but it is, obviously, not an entirely effective one. Vaccines are only partially effective for the control of skin infections and have had little or no success in the treatment of periodontal diseases. Antibiotics if they can be gotten to the site of infection may be of use. Their position in the treatment of periodontal disease does not yet appear established. Glandular secretions and the structural characteristics of the mucous membrane are of critical importance in resisting invasion by bacteria. Saliva has antibacterial properties. Surgical extirpation of the salivary glands in the albino rat has been shown to induce the development of a filth type of periodontal disease.[4]

Cornified cells are also protective. Judicious use of the tooth brush may help to increase the protective horn barrier of the epidermal cells against infection. Local factors such as the impaction of food, third molar flaps, improper tooth contours, and overhanging margins of crowns and fillings tend to weaken the tissue defenses. It must always be kept in mind that resistance to periodontal infection depends at least as much on intrinsic factors as on extrinsic ones.

Another pathogenic micro-organism which "causes" a disease is Treponema pallidum. These spiral organisms are transmitted by contact and appear to be capable of making their way through the intact mucous membrane. The site of inoculation breaks down in two or three weeks to form a relatively painless ulcer or hard chancre. A few weeks later the secondary rash and mucous patches appear. During this period, the blood and tissues and the local lesions are swarming with the micro-organisms. The patient is highly infectious to others, yet he is ordinarily not seriously ill and may be entirely unaware that he is diseased. The lesions appear to heal completely and no overt evidence of the disease may remain. Years later gummata, cardiovascular or central nervous system lesions may appear. The micro-organisms then are scarce and difficult to demonstrate. If it were not for the Wassermann and other serologic tests the connection between late and early lesions of syphilis might not be recognizable. It is possible that other diseases exist, for which we have no present means of detection, which manifest themselves by degenerative change years after the primary period of infection has passed.

One group of the spirochetal organisms of Vincent's disease morphologically closely resembles the Treponema pallidum of syphilis. Vincent's disease has proven extremely difficult to transmit to humans or to experimental animals except by a mixed infection with at least four types of organisms. Many investigators have come to the conclusion that Vincent's disease is not infectious because of the presence of Vincent's organisms in clinically healthy mouths and because of the difficulty of transmitting the disease. Yet the dramatic subsidence of signs and symptoms of the acute phase of the disease upon treatment with antibiotics indicates that the micro-organisms, which concomitantly tend to disappear, probably are etiologically significant.[5] The role of Vincent's organisms in the initiation and progress of periodontal disease should be reinvestigated.

Over 700 parasitic micro-organisms have been associated with infectious diseases. Plant forms, bacteria and molds produce about 200 of the infectious diseases. A table of living agents responsible for disease, after Gay et al.,[6] is given in Chart 1.

It is obvious that certain etiological agents were discovered a long time ago, while others have been identified only re-

Living Agents Responsible for Disease, In Order of Total Numbers

	Total Number	*First Organism Described*	*Discoverer*	*Date*
1. Bacteria	123	Actinomyces bovis	Langenbeck	1845
2. Helminths	95	Fasciola hepatica	Jehan de Brie	1379
3. Fungi	81	Botrytis bassiana	Bassi	1837
4. Arthropods	71	Phthirius inguinalis	Redi	1668
5. Protozoa	56	Eimeria stiedae	Hoke	1839
6. Chlamydozoa	25	Guarnieri bodies	Guarnieri	1892
7. Spirochetes	13	Bor. recurrentis	Obermeier	1873
8. Rickettsiae	5	Dermacentroxenus rickettsi	Ricketts	1909

CHART ONE

cently. Therefore, the possibility of discovering specific, infectious, etiologic agents for periodontal diseases has not been exhausted.

Non-infective micro-organisms also play a role in the etiology of periodontal disease. They are associated with clinical conditions usually described as poor oral hygiene. Two types may be considered, although they usually exist together and reinforce each other. First, putrefactive organisms associated with the accumulation of food and other soft debris are found about the necks of the teeth. Irritative decomposition products may cause inflammatory changes in the neighboring gingival tissues. Second, calcified deposits occur above the gingival crest (supragingival calculus) and between the epithelial lining of the gingival sulcus and the tooth surface (subgingival calculus). Micro-organisms of the actinomyces group and others serve as a matrix in which calculus deposition occurs. The organisms also contain phosphatases, enzymes which promote concentration of calculus salts and their deposition.

The injury producing agents we have considered so far are living organisms which directly or indirectly interfere with the well-being of the host. This they do by producing poisonous substances (chemical damage) or by mechanical injury (physical damage). Clearly, damage may be produced also by non-living agents which again damage the tissues by chemical or physical means. Once calculus has formed it acts as a physical irritant and the injury is continuous. A first principle of treatment is the removal of calculus in order that the processes of inflammation, the immediate response to injury, may be followed by processes of repair. The initiation of the periodontal lesion, and the events leading up to the deposition of calculus within the gingival sulcus, remain largely unknown, and certainly should be thoroughly investigated.

The impaction of food, improper tooth cleaning practices, and faulty dental restorations may cause local injury to the periodontal tissues with resulting inflammatory and reparative processes. Recognition of the injury producing condition and its correction, ordinarily, is followed by a prompt return of the periodontal tissues to a healthy state. The likelihood of an untreated traumatic gingivitis eventually progressing to advanced periodontal disease appears to be reasonably great. The factors of bacterial infection and calculus formation are important if not essential complicating factors in this process.

Another injury producing mechanism which plays at least an accessory etiologic role in the development of periodontal disease is traumatic occlusion. Traumatic occlusion is recognized after the damage to the periodontium has occurred. The causative factors of malocclusion are inferred from an examination of the location and extent of the periodontal lesions. It is

not possible at the present stage of our knowledge to examine the muscular complex, habits of mastication, position and inclination of teeth, the cusp forms, or the degree of overbite and interlocking of the teeth and to predict with assurance the condition of the periodontal tissues. Once periodontal disease has been established, it is frequently possible to recognize predisposing factors concerned with occlusal dysfunction. Correction of malocclusions is recognized as an important adjunct to the treatment of periodontal disease. This is a special application of the general principle that injured and diseased tissues repair themselves most rapidly and effectively when put at rest.

The length of the tooth root as compared to the crown and the structure of the periodontal membrane and alveolar bone are of importance in withstanding the functional stresses of occlusion and mastication. Teeth which have drifted from their normal position, particularly after the extraction of neighboring teeth, are usually tilted at abnormal angles and masticatory forces are applied in non-physiologic directions. They also lack the mechanical support of adjacent teeth. Such teeth are particularly susceptible to the development of periodontal disease, though again the association is by no means invariable.

The reasons why traumatic occlusion, poor oral hygiene or other injurious factors lead to periodontal disease in one patient and not in another carry us into a very complex field of discussion. We may use such terms as heredity or constitution, resistance, active and passive immunity, metabolic disorders, nutritional and endocrine disturbances or imbalances. Here we are concerned with the normal functioning of the cells, tissues and organs of the body and with the adaptive responses by which they meet adverse conditions, successfully in health and unsuccessfully in disease.

Genetics is a highly advanced field of biology which approaches mathematical exactness and predictability to a far greater degree than many scientific disciplines which are utilized by dentistry and medicine. Certain human diseases, hemophilia for example, are inherited in a definite pattern. A familial pattern is reasonably clear in several metabolic diseases including diabetes mellitus. Some types of baldness are hereditary. It appears possible that the tendency to periodontal disease may also be familial.

Nutritional inadequacies are now universally recognized as a cause of specific disease states. Here we are dealing with specific chemical compounds which are essential for the normal functioning of the cells. In the case of the vitamins, organic compounds in small quantities are essential. Usually, if not always, they serve as parts of cell enzyme systems. We come close here to the border between extrinsic and intrinsic etiologic agents. The vitamins offer a chemical dissecting tool to help us unravel the specific requirements of particular cells and tissues.

In periodontal disease, vitamin A has been shown to be concerned with abnormal cornification of epitheliums and probably also with osteogenesis. Vitamin C governs the production of collagen, including periodontal fibers and bone matrix, and vitamin D is essential for normal calcification of the dental tissues as well as of bone.[7,8]

Nutritional inadequacy may be primary or secondary. Primary deficiencies may be found in starvation, alcoholism, and medical or self-imposed dietary regimes. Secondary deficiencies may occur as the result of intestinal disease with inadequate absorption; as the result of liver disease with defective metabolism; or from increased demands in fever, hyperthyroidism and pregnancy.

Many periodontists are aware that primary nutritional inadequacies predispose to the development of periodontal disease or interfere with the proper response of the tissues to therapy. Nutritional deficiencies secondary to increased demands or inadequate absorption are less well recognized. In infections, fevers and hyperthyroidism

conditions where the basal metabolic rate is increased, the need for vitamins as well as for proteins is increased. The absorption of fat soluble vitamins from the intestinal tract may be interfered with by the use of mineral oils taken immediately after eating. Absorption may be inadequate also in sprue, ulcerative colitis or other inflammatory conditions or after surgical resection of part of the intestinal tract. It is clear that the subject of nutrition is more complex than merely the selection of diet.

The endocrine glands come still closer to the internal machinery of the body, yet they may also be affected by external agents. The thyroid gland which governs metabolism may have its effectiveness neutralized by the drug thiouracil, for example. The complex interrelations of the endocrine glands, their effects upon cells, tissues and organs and the results of stress or trauma are much too vast a subject to attempt to discuss here and therefore, reference is made to the works of Albright,[9] Selye[10] and others.

SUMMARY

Etiology, the study of causes, is one of the three major phases of pathology, the study of disease. Etiologic agents produce injuries, these in turn cause responses to injury or processes and the results of the processes constitute disease itself. Etiologic agents are very numerous. They may be living, self-producing organisms or may be physical or chemical in nature. Intrinsic as well as extrinsic causes of disease are recognized. Processes, on the other hand, are relatively few in number and are essentially submissive, reactive and adaptive in character.

A variety of infectious micro-organisms appear to be associated non-specifically with the initiation and progress of periodontal disease. Non-pathogenic micro-organisms are apparently also of importance in the production of irritating products, including calculus, which in turn act as mechanical and physical injury producing agents. Traumatic occlusion is a localizing and predisposing factor in the etiology of periodontal disease. Faulty dental restorations and injurious tooth cleansing practices also predispose to the development of periodontal disease.

Intrinsic factors including heredity, nutritional and endocrine status play an important role in determining the resistance or susceptibility to periodontal disease.

No specific etiologic agent for periodontal disease or specific therapeutic measure is apparent at the present time, but it is possible that future research will reveal such agent or agents and indicate more rational and effective therapy than is at present employed.

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Report of a Study on the Relationship of Personality Variables to Periodontal Conditions*

by Lt. John H. Manhold, d.c., U.S.N.,† *School of Aviation Medicine, Pensacola, Fla.*

In an earlier preliminary report,[4] Manhold and Manhold presented the first results of a statistically significant nature to reinforce oral psychosomatic theories. They found a positive correlation between dental caries and two personality traits in subjects who could be classified as normal from a psychiatric standpoint. These results gave credence to assumptions made by Mellars and Herms,[5] Miller and Firestone,[6] Briggs,[2] Gill,[3] and others that oral pathological conditions as well as bodily dysfunctions could have a psychosomatic basis.

The objective of this part of the study is to discover whether a similar correlation appears to exist between periodontal disease and psychological factors in essentially normal people.

The systems of measurement employed were the *Personality Inventory* by Robert G. Bernreuter[1] and a complete clinical and roentgenographical survey. The *Inventory* was previously selected because it permits the measurement of several different traits of personality simultaneously without obviating the nature of the qualities under study to either the examiner or the examinee. It purports to measure neurotic tendency, self-sufficiency, introversion-extroversion, dominance-submission, confidence in one's self and sociability.

A group of fifty subjects was used in this study. It was composed of the first fifty persons being processed through the United States Naval Training Center, Great Lakes, Illinois, on their return to active duty who had periodontal disease. The group included thirty-eight men and twelve women. Patients with gross malocclusion or other similar contributory oral defects were excluded. The age, sex, and education of each person were recorded along with a code number. The age range of the subjects was from seventeen to forty with a median of twenty-five and a mean of twenty-seven. Code numbers were used to overcome the subject's possible hesitancy

*Opinions or conclusions contained in this report are those of the author. They are not to be construed as necessarily reflecting the view or the endorsement of the Navy Department.

†School of Aviation Medicine.

PERCENTILE EVALUATIONS

IN RELATION TO PERIODONTAL CONDITIONS

TABLE 1

	STANDARD MEAN	DEVIATION	MEDIAN	RANGE	NUMBER OF PATIENTS 59 and BELOW 1/	60 and ABOVE	TOTAL
NEUROTIC TENDENCIES							
CLASS I 2/	28.7	24.5	12.0	0-97	21	2	23
CLASS II 3/	65.8	18.1	74.0	16-100	12	15	27
INTROVERSION-EXTROVERSION							
CLASS I 2/	25.8	23.5	18.0	1-89	21	2	23
CLASS II 3/	62.8	27.8	66.0	7-100	11	16	27

1/ 59th PERCENTILE (THIRD QUINTILE) - THE UPPER LEVEL OF NORMALCY

2/ CLASS I MILD PERIODONTAL DESTRUCTION

3/ CLASS II SEVERE PERIODONTAL DESTRUCTION

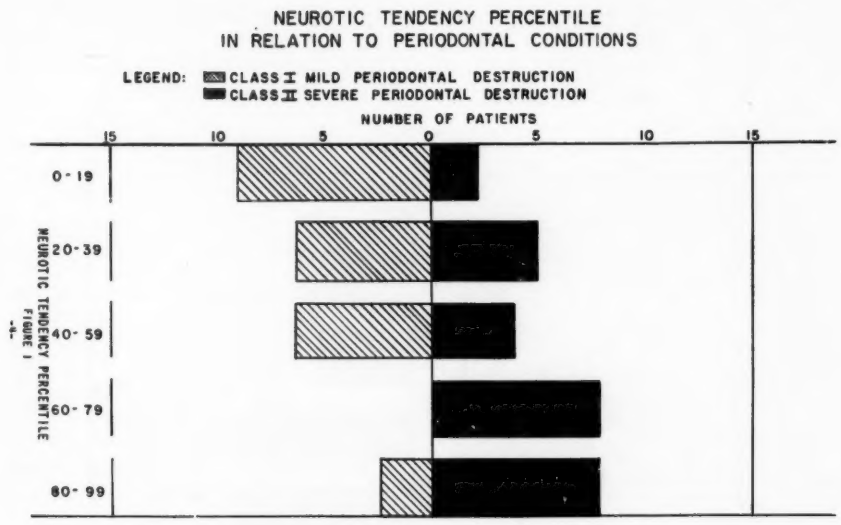

FIGURE 1

to truthfully answer the psychological questions. The personality tests were scored for all six traits with the aid of Bernreuter standard scoring sheets, and percentiles were found on the standardized scale. The extensiveness of the periodontal pathology was determined by a complete clinical and roentgenographical survey. For the purpose of statistical analysis, cases were categorized as accurately as possible into two classes according to the severity of the condition.

As in the caries study, the only personality traits which correlated significantly with periodontal pathology were the measures of neurotic tendency and introversion-extroversion. On the Personality Inventory the third quintile, whose upper limit is the fifty-ninth percentile, represents the upper level of normalcy formerly established and was again employed as the line of demarcation. Persons exhibiting the greater neurotic tendency, i.e., those *above* the third quintile, presented the greater number of severe or Class II periodontal conditions. Those persons scoring below this level appeared to be mostly of the Class I variety. Analogous results were gained from the introversion tendency study—in general the more introvertive subjects exhibited the more severe periodontal conditions. These results are pictured in Table I and Figures 1 and 2.

In terms of percentiles the mean value for neurotic tendency was 28.7 for Class I cases of periodontal destruction and 65.8 for Class II cases. The respective medians were 12.0 and 74.0. The mean introversion-extroversion percentile for Class I cases was 25.8 while that of persons with Class II conditions was 62.8. The medians were 18.0 and 66.0 respectively. Age did not appear to be an important factor.

The critical ratios comparing the personality trait scores of subjects with Class I Periodontal destruction against personality trait scores of subjects with Class II destruction yielded values significant at the .003 level. In other words, there are fewer than three chances in one thousand of this distribution being due to chance.

Classification of periodontal cases is admittedly open to controversy, but is a necessary procedure to allow statistical analysis. However, the method of classification employed by the author is less con-

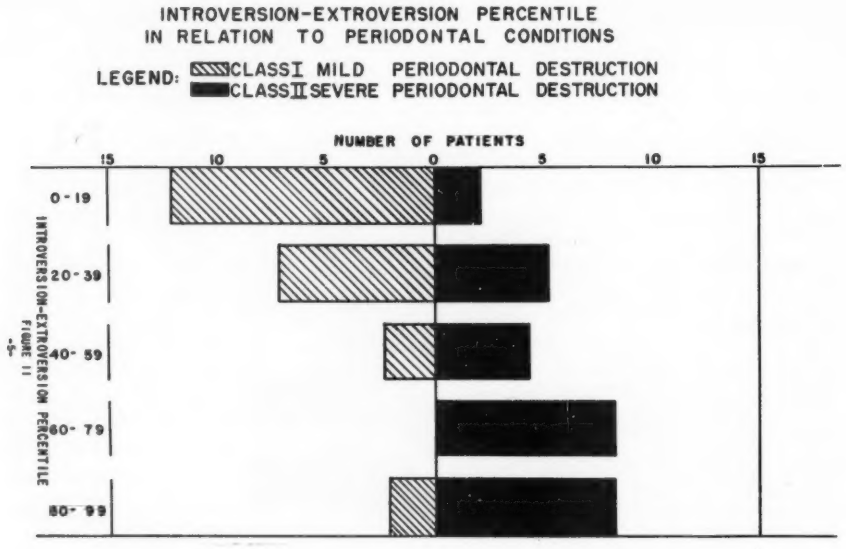

FIGURE 11

troversial than any of the other methods of choice used by periodontists today. It is a rating which assumes only that one pathological periodontal condition can be more severe than another.

Borderline cases can be contested, but these criteria, together with the number of cases which fall definitely into the specific categories, appear to make prolonged arguments on these ratings pointless. Furthermore, the mean and median values for neurotic tendency and introversion-extroversion percentiles also exhibited an excellent relationship with the respective classes of destruction.

The Bernreuter Personality Inventory, from a biometrician's standpoint, is not an exact measurement of personality. It is one of many inventories in use today by psychologists which purportedly measures traits of personality as divorced from intelligence. It is advantageous because it permits a rapid, uncomplicated measurement of these traits. Its prime limitation, as discerned by further studies[7] by the author using this test in conjunction with other instruments of measurement, is that it does not probe deeply enough into the underlying strata of a subject's personality. If the person has been "psychologically screened," or is in any way familiar with testing procedures, his answers will often not reflect his true personality. The subjects used in this project were, to the best of the author's knowledge, not "test wise," however.

The primary importance of this study lies in the fact that a relationship appears to exist between some traits of personality and pathological periodontal conditions. It appears all the more important because of recent results of another nature reported by Manhold.[8] In a follow-up study on his preliminary report of a relationship between personality and dental caries, he again found a statistically significant correlation between the two variables using a larger population and more varied tests. The underlying psychological meaning of his results is as yet obscure, but the important fact is that a definite relationship was demonstrated. Now, if positive results were obtained of a relationship between the pathology of hard tissue and personality traits, it would certainly seem very

plausible that a relationship could exist between personality factors and soft tissue pathology. Just such a relationship was demonstrated here. In the light of these findings, then, and with the positive results from studies of the influence of personality factors on hard tissue pathology, it would appear that these results are highly significant, and that similarly significant results could be expected from further work in this vein. It is suggested that projects of this nature be undertaken with larger groups and more varied tests.

ACKNOWLEDGEMENT

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ACADEMY OF PERIODONTOLOGY ELECTS NEW OFFICERS

The January 1954 issue of the Journal of Periodontology will include a detailed report of the very successful 37th Annual Meeting of the Academy of Periodontology which was held in Cleveland September 24, 25 and 26, 1953. Committee chairmen are urged to send their reports to Secretary Chamberlain at once if they have not already done so.

Officers for 1953-54 are:

President: Harry Lyons, Medical College of Virginia, Richmond, Virginia.
Pres.-Elect: Harold Ray, University of California College of Dentistry, San Francisco, California.
Secretary: Clarke Chamberlain, 115 North St., Peoria, Illinois.
Treasurer: James Aiguier, 1116 Medical Arts Bldg., Philadelphia 2, Pennsylvania.

New Council members are:

Harry Wood, 1265 David Whitney Bldg., Detroit, Michigan.
Mildred Dickerson, 1726 Eye St., N.W., Washington 6, D. C.

An Evaluation of Factors Determining Prognosis in Inflammatory and Retrogressive Periodontal Disease: A Series

by RICHARD WILLIAM SLATTEN,* D.D.S., *Richmond, Va.*

A FACTOR of major importance in the consideration of any type of disease is the determination of its prognosis. Prognosis may be defined as "a forecast of the probable result of an attack of disease: the prospect as to recovery from a disease afforded by the nature and symptoms of the case (at hand)."[44]

"Prognosis" comes to us from the Greek word meaning "foreknowledge." Therefore, it follows etymologically that prognoses in disease, and periodontal pathosis is no exception, are basically concerned with a foreknowledge of the course of disease. Moreover, suffice it to say that the matter of evolving a prognosis is directly dependent on an accurate diagnosis. A detailed reflection on methods of diagnosis is beyond the scope of this paper, but it can be appreciated that one cannot be discussed without an assumption of the other.

A "good" or "favorable" prognosis may be interpreted as one which predicts a satisfactory return to health. An inference may be taken that the outlook for maintenance of health and function is decidedly unsatisfactory when reference is made to a "poor" or "unfavorable" prognosis. The term "hopeless prognosis" is reserved here for indicating the complete improbability that natural masticatory structures will or should be retained. The use of the terms "negative" and "positive" as applied to prognosis will be avoided so as not to promote ambiguity.

Since a correct diagnosis is the groundwork on which the outcome of disease is predicted it would seem in order to commence with a discussion of the over-all prognosis of the several broad classifications of periodontal disease.

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Lyons[26] has classified periodontal disease in three general categories, i.e. inflammatory, retrogressive, and neoplastic. The group of *inflammatory periodontal diseases* includes the gingivides, acute and chronic, and the periodontides, simplex and complex. The *retrogressive periodontal diseases* include the periodontoses whose nature may be atrophic, hyperfunctional, or primarily of systemic origin (periodontosis gravis). The category of *neoplastic periodontal diseases* includes all tumors of the gingiva and periodontium, both benign and malignant. This paper deals with the gingivides; a subsequent paper will consider the periodontides, and the periodontoses.

The importance of the consideration of the many types of gingivitis and their respective prognoses should not be minimized. Cases of gingival inflammation in which the prognosis is poor condemn the patient, in the long run, to secondary disorders of the entire periodontium.

The subject of gingival inflammatory changes should be approached with a knowledge of ulatrophy itself. Ulatrophic changes, in the strictest sense, are not characterized by inflammation but it should be remembered that they may lead to or become complicated by inflammation.

By nature, gingivides may fall into several diverse classes. From the aspect of duration they may be classified as acute or chronic and in general it may be said that inflammations of an acute nature are more amenable to treatment than chronic ones. In addition, both acute and chronic inflammations of the gingivae may be simple or purulent (exudative), with chronic ones subject to several additional inflammatory states (see below). For the most part, inflammations of a simple nature offer the better prognosis. However, it would be

both difficult and misleading to attempt a prognosis on such narrow limitations as these, for further type-breakdowns of both acute and chronic gingivides can be identified.

Ulcerative Gingivitis includes a broad scope of disorders. It may be seen preceding, in conjunction with, or complicating (general) ulcerative stomatides. The literature, medical and dental, teems with a variety of nomenclatures, much of which we find at cross-purposes.

A great number of gingival ulcerations should probably, at this time, be classified as non-specific. Several special infections, e.g. syphilis and tuberculosis, etc., may give rise to ulcerative conditions on the gingivae but their treatment and the determination of their prognosis lie outside the field of dentistry. (They would, of course, find a place of major importance in a discussion of differential diagnosis).

Ulceration is a free surface lesion, suppurative, necrotic, or traumatic in nature. A classical example of ulceration, and probably one of the most frequently seen ulcerative lesions of the gingiva, is the *aphthous ulcer.* According to McCall[27] it appears most commonly in the muco-buccal fold or on the lingual side of the mandibular alveolar ridge at the junction of the alveolar mucosa and the floor of the mouth in which location its etiology is attributed to systemic influences such as slight fever, gastric derangement, menstruation, psychic disturbances, and allergies. A similar lesion may appear elsewhere in the mouth whose causation is traumatic in origin. It can occur singly or multiply. In cases of *recurrent aphthae* the gingivae are seldom affected. Whatever the location, severity, or etiology, aphthous lesions appear always to run a subacute course and resolve themselves, healing taking place in from one to two weeks.

Another ulcerative condition, not uncommon to the mouth, has been attributed to the herpes simplex virus. Scott et al.[33] indicate that the majority of persons experience their first infection with the herpes simplex virus during infancy or early childhood. The primary attack may go unnoticed although it may produce a clinical picture of fever, irritability, soreness of the mouth, red swollen gums, oral fetor, and regional lymphadenopathy. In about 1 per cent of the cases it may be serious or even fatal. He goes on: "The clinical entity of *acute herpetic gingivostomatitis* is the commonest primary infection in childhood. It is marked by an acute febrile illness. The common adult manifestation is *recurrent herpes labialis* or 'fever blisters.' These attacks are considered to be caused by reactivation of the latent herpes virus in the tissues. However, there is no laboratory evidence to support this." Dodd and Ruchman[13] say the herpes simplex virus is not the etiological agent in recurrent stomatitis (recurrent herpes labialis) although they base their statement solely on the fact that they were not able to secure the virus from aphthous lesions. Scott et al. continue: "Primary herpetic infection in adults is rare. The disease appears to be self-limiting and conclusions as to benefit of various therapeutic agents must be based on this knowledge." Recent evidence points to the fact that aureomycin seems to alter sharply the course of acute herpetic gingiviostomatitis, often within a period of forty-eight hours.

A rather common form of gingival pathosis, and the one which is probably of the greatest concern to the dentist directly, has been known over the years under the varying titles of *Vincent's Infection, ulceromembranous gingivitis, trench mouth,* and *necrotizing ulcerative gingivitis.* The literature abounds with descriptions of its syndrome and varying prognoses have been forecast for mouths afflicted with it. It has probably received more attention in discussions of the empirical use of drugs in dentistry than any other condition. It has been studied exhaustively over the years by various research workers. It still remains imperfectly understood.

In spite of the faults in our knowledge

concerning *necrotizing ulcerative gingivitis* great strides have been made toward a perfect understanding of its entire picture. First, and foremost, the course of the disease has been established and a full understanding of this course is necessary in order to outline its prognosis.

Hill[21] states that "necrotizing ulcerative gingivitis in the mouth is a variable process, its differing phases depending upon the type and activity of the infective organism, the state of oral sanitation, and the health and resistance of the patient. It may be acute, subacute, or chronic and it may be recurrent." It may be widely distributed in scope or localized in a few areas or even in one single area. It simulates other true periodontal diseases in that it occurs only in mouths in which teeth are present.

From a clinical point of view the disease is probably most often recognized in its acute stage. A list of its "classical symptoms" may be found in almost every good treatise on oral pathology. It should be realized, however, that many of these symptoms may vary as widely as the multifold phases of the disease itself and careful scrutiny of the mouth is often necessary before a diagnosis can be made. This is particularly true in chronic and subacute cases and in the periods of remission which, alternating with the periods of exacerbation, form the typical course of the uncontrolled case.

Destruction of the interdental papillae, resulting in a distinctive, "punched-out" deformity, is pathognomonic of necrotizing ulcerative gingivitis. It appears in all the panoramic phases of the disease with the possible exception of the very early stages of *initial* (acute) onset. (The absence of damage to the papilla during that particular period, which is extremely short, should not hinder diagnosis for at that time the other classical symptoms, oral and systemic, are at their height). Destruction of the interdental papilla is not only the disease's most distinctive feature but in addition is the basic consideration on which the rationale of treatment has been founded and on which prognoses are based. A consideration of the fate of the gingival papilla supercedes all other considerations of gingival alterations. The detrimental consequences which arise following its loss encompass a tremendous range of ensuing pathosis. The importance of its conservation wherever possible cannot be over-emphasized.

The control and rapid suppression of the acute oral symptoms of necrotizing ulcerative gingivitis are no longer considered a problem. The multitude of drugs which have been used and recommended for the suppression of the symptoms of the disease have failed completely to accomplish the foremost principal by which therapeutic measures must be judged, i. e. they have failed to alter favorably the course of the disease; they have, in brief, failed to preserve the gingival papilla. The use of the antibiotics has received wide acclaim in the literature and their use as adjuncts, particularly for alleviation of acute systemic symptoms (when these are present in accentuated form) is not to be overlooked. But the prognosis of this disease entity itself (if it may be so termed) as well as the outlook for the deeper periodontal structures over a long range period, must be based on a consideration for conservation of the interdental papillae. Mutilation of this important anatomical structure should always be evaluated as a crippling deformity.

The rationale of the treatment of necrotizing ulcerative gingivitis will not be discussed here. If scientifically founded treatment is instituted early enough, conservation of the papillae is highly probable. Troublesome problems must be faced where sloughing of the papillae has already occurred.

The deformity resulting from sloughing of the gingival papilla poses two distinct problems for the clinician. Of immediate concern is the resulting harbor the concave nature of whose floor encourages food impaction, calculus formation and collection, and bacterial growth. Such impaction,

formation, and growth await only the unknown, but highly important, "lowered-tissue-resistance" factor's appearance and recurrence of the disease may follow. Secondly, the clinician is concerned, when dealing with mouths where sloughing has occurred, with the susceptibility of such areas to deeper periodontal pathosis. Areas of sloughing may already have involved partially or wholly the alveolar crest and transeptal fibers of the periodontal membrane, both of which are concerned in the pathogenesis of incipient simplex periodontitis.

Such sequelae need not be considered as invariable, of course. Certain factors will tend to alter final developments. The location and extent of the sloughed area, the general health of the patient (which directly affects the resistance of the oral tissues), the interest in hygiene which the patient manifests, and the general picture of tooth alignment and occlusal relationships should all be weighed carefully before making the patient aware of what he may or may not expect to experience dentally and orally in the future. Moreover, tissue response to treatment should influence the prognosis in these cases. Young, healthy tissues may be expected to exhibit regenerative powers superior to those of aged or debilitated tissues. The evidence of even partial regeneration of the papilla, more likely to occur in the anterior regions accessible to cleaning, should be counted as favoring the maintenance of health.

Clinical procedures which direct attention at conservation of the interdental papillae are based on very fundamental criteria. The high incidence of simplex periodontides exhibiting defects bearing the unmistakable stamp of necrotizing processes has far-reaching implications. The existence of such defects should caution the wise against overly optimistic outlooks.

The gingiva is subject to several additional inflammatory states at a chronic level. They may be grouped and considered, according to tissue change, as: *hyperplastic*, *desquamative*, and *pigmented*.

Hyperplastic tissue change includes all those changes in which there is an excessive formation of tissue or an increase in the size of a tissue (or organ) owing to an increase in the number of cells. Such changes appear to take place readily in the gingivae and will do so in response to a wide diversification of etiological agents. Hyperplasia may develop in cases characterized by poor or inadequate oral sanitation, trauma, or endocrinopathies. It may result in certain hemopathic disorders, or as a response to certain drugs, and, according to some, as an allergic manifestation. It quite possibly results from etiological agents not now identified. Gingival disease probably most often has a complex causation. It is, therefore, not always possible to point to any one causative factor and label it the one most culpable. That is why the classification of periodontal disease on a basis of etiology is prone to place a stumbling-block in the path of the student who is attempting to gain clear insight into the subject. For example, a case of periodontal disease, hyperplastic in nature, may have a background of diabetes, present a picture of gross oral neglect, and be further complicated by trauma.

POOR ORAL SANITATION

Poor or inadequate oral sanitation has long been associated with inflammatory reactions in the gingival tissues. Miller[28] classifies this type of gingivitis as *indolent* and reports that the effects of a non-detergent diet should be considered in its causation. Smoking, in relation to gingival disease, may be included as an etiological entity along with other unhygienic practices. Smoke might be considered as an extraneous substance capable, under certain conditions, of giving rise to irritation. The exact manner in which smoke affects the gingiva bears further investigation although smoking itself could even now not be considered salubrious.

As a general rule, incomplete or poor oral sanitation will manifest itself readily in the majority of mouths. The gingiva

may show a variety of reactions to food accumulation though generally it appears to arouse only mild inflammatory reactions in the marginal gingivae and tips of the interproximal papillae. Neglect, so often evident in mouths characterized by hyperplasia, consequently falls under suspicion as an etiological agent of importance, especially in the light of the clinical impression that vast numbers of cases which resolve almost completely, but never quite entirely (under treatment where home care is never maximum), proceed to complete resolution when the quality of the home care is raised.

The prognosis of gingivitis of such a nature is good, all other things being equal, if patient cooperation is obtained. Such cooperation is not always possible, of course. Poor alignment of teeth, impaired manual dexterity, central nervous system involvements, and adverse psychic manifestations may be present to prevent patient participation in an adequate home care regime. Such problems have a very definite adverse effect on prognosis and when they exist a complete return to health will probably never be accomplished; in fact, conditions ultimately go from bad to worse in such instances.

Some gingival tissues show definite response to trauma albeit clinically the gingiva in such cases does not always produce a classical picture of inflammation. Trauma may be inflicted on the gingiva *directly* as in food impaction, or by toothbrushes, toothpicks, and other cleaning devices, or by direct trauma of incisal edges or occlusal surfaces of teeth themselves. The gingiva may also react to trauma as it is transmitted *indirectly* through a tooth, being responsible, according to some, for the characteristic "traumatic festoons" of the gingiva, often referred to as "McCall's Festoons." These teeth may be in a traumatic relationship with an antagonist or they may be agents abused by the patient in some biting habit. Nail, pencil, lip, tongue and thread biting, bobby pin opening, etc., have come under surveillance and been condemned. Sorrin[34] has made an extensive classification of habit as an etiological factor in periodontal disease.

NUTRITION

The body may be considered as a mechanism which is constantly striving to maintain that harmonious status we term "health."[8] Without proper nutrition individual cells cannot be expected to maintain, defend, and replace themselves and ultimate tissue breakdown or change of one type or another is inevitable. Nutrition is an extremely complex physiological process and diet is certainly an indispensable part of it. A balanced dietary program does not insure adequate nutrition but an absence of certain food elements from the diet certainly precludes the possibility of a suitable nutrition.

The gingiva often becomes hyperplastic in states of defective nutrition and the concept now held in highest regard concerning states of avitaminosis lays stress on the importance of multiple vitamin deficiencies in the causation of tissue disease. Spies et al.,[35] among others, propose this concept and the work of Sud[38] appears to be in accord with it. Sud goes on to say that the existence of local factors exaggerates the effects of nutritional deficiencies in the development of gingivitis.

In general it would seem that the prognosis of gingival disease whose etiology is concerned with dietary deficiencies is good providing local exciting factors can be eliminated and a balanced dietary program provided. If a basic nutritional dysfunction is identified or suspected the outlook for a return to health may not be so bright. The diverse causes of failure to assimilate the necessary food elements are not always controllable and, when this is the case, predispose the individual to a continued divergence from a state of health.

Various dysfunctions or states of imbalance in the endocrine system present not infrequent changes in the priodontium. The gingiva is susceptible to such dysfunctions or derangements in balance and

alterations are definitely known to result.

The gingival changes associated both with menstruation and with pregnancy are concerned, at least indirectly, with the variations in estrogen production and levels. Estrogen levels are known to fluctuate widely in the normal menstrual cycle and even more bizarre vacillations may exist in those women who experience irregular mences. In puberty, when the cycle is subject to variation before a definite pattern has been established, gingival changes have been noted. The dentist should be cognizant of the relationship of the gingiva to these phenomena and the physician should be asked to cooperate in controlling such endocrine alterations.

Curves showing estrogen levels during pregnancy follow a general upward path. By the middle of the first trimester estrogen levels are higher than at any time in menstruation. Progressively higher levels of estrogen are produced and maintained through the thirty-sixth week of pregnancy and reach their peak shortly after that time, falling off markedly just prior to labor.[7]

In certain phases of pregnancy and menstruation the epithelium of portions of the genito-urinary tract and the ovary are characterized by vigorous inflammatory reactions.

Cowdry[12] points out that "after a consideration of the changes in the various parts of the female reproductive tract and the ovary, one is impressed by the striking similarity between these reactions and the ordinary inflammatory processes which follow acute irritation or infection. Few, if any, normal physiological changes in the mammalian body cause structural reactions so typically the same as those usually recognized under pathological conditions as does the process of ovulation. The estral reactions emphasize the fact that inflammatory processes are, after all, really physiological; that is, functional tissue responses for protective and regenerative purposes."

Since the oral mucosa and the epithelium of the reproductive tract and organs are similar (and, perhaps, associated) structures, it may be that some gingival changes are at times simply a reflection of reproductive-epithelial changes. It is possible that the gingival changes often seen in pregnancy would not necessarily be clinically manifest if there were an absence of local irritating factors in the gingiva. On the other hand, the gingiva, characterized by its dense capillary bed, may be so constituted as to express inflammatory reactions even under almost ideal conditions. Both the mucous membranes of the cheek and the ventral surfaces of the tongue are subject to changes in their pattern of cornification, similar to those in the gingiva, under the influence of variations in the estrogen level according to Iusem.[24] These structures might logically be expected to show changes similar to those in the gingiva if local irritants of a similar nature were brought to bear on them, or if their anatomical structure had the same inherent peculiarities as the gingiva. Ziskin's work on the effects of testosterone propionate and the estrogenic hormone seems pertinent here. He reports that both the male and female sex hormones are tissue stimulators of the non-specific type. Both produce hyperkeratinization. Hyperplasia is evident in the keratin layer of the epithelium and is especially marked in the corium of the connective tissue. Of the latter he says: ". . . definite changes take place in the corium. Hyperplasia is marked. Large collagenous bundles comprise the greater portion of the tissue. A moderate increase is noted in the number of active fibroblasts scattered throughout. Many fibrocytes, apparently devoid of activity, are also seen between bundles. The extensiveness of the collagenous bundles accounts for the homogenous, hyaline-like appearance of the connective tissue and its increase in thickness. Vascularity is slightly more pronounced. Edema is present in isolated areas."[42]

The gingiva of individuals in puberty may often present a baffling problem. To begin with, individuals of that age range are, as a rule, difficult patients to approach.

The amount of cooperation they give in home care and in following their physician's advice is variable and uncertain. If extensive caries is present the problem is obviously even more complicated for response to treatment is slower in the face of traumatizing operative procedures. However, if the patient receives periodic periodontal treatment and results are satisfactory enough to prevent deeper periodontal involvements from occurring there is no reason to suppose the prognosis is not good providing any existing endocrine disbalance is controlled or ultimately resolves itself.

The gingiva in pregnancy may present a troublesome clinical entity but the rationale for its control has been satisfactorily established and suffices in the majority of cases. Good periodontal therapy, of a conservative nature, will serve to keep gingival reactions to a minimum although a return to a complete state of health probably will not follow until pregnancy has terminated. The prognosis for all structures concerned is good when treatment is instituted early and is continuous through the last trimester, if such appears to be indicated. So called "pregnancy tumors" should probably be treated conservatively unless they grow to unusual proportions since surgical intervention may result in deformities of the margin and papilla.

An alteration in the endocrine system is apparent in the diabetic patient. The literature has described numerous gingival changes in diabetes mellitus labeled "characteristic." It is probably amiss to attempt the classification of any group of gingival alterations as characteristic of the diabetic patient and the recent work of Ray and Orban[20] seems to substantiate this. They report the following:

I. Clinical aspects of the diabetic gingiva:

a. no diabetic patients were free from gingivitis.

b. there was no correlation between the control of diabetes and the severity of the gingivitis, however, all cases of severe gingivitis were seen in patients with poor control.

c. the incidence of a smooth epithelial surface and an absence of stippling increased with the severity of the gingivitis and was more noticeable in diabetics than in controls.

II. Histological aspects of the diabetic gingiva:

a. histologic study failed to show changes characteristic for the diabetic gingiva.

b. intranuclear vacuolation of basal layer epithelial cells and increase in intensity of the inflammation was seen oftener in diabetics than controls.

c. there was no difference in the glycogen content of the tissues of diabetics and controls.

Such findings seem to indicate that the diabetic patient is more subject to gingivitis than the nondiabetic and that the gingivitis present will likely be of a more severe nature than that present in the mouth of a nondiabetic patient. Information of this nature will lead practitioners to view the gingivae of diabetic patients with concern and to establish prognoses of an unfavorable nature.

Such patients should be treated from a conservative standpoint and the dentist, if he is to anticipate favorable results of any degree, should insist that the patient be under the treatment of a physician.

The hemopathic disorders of the greatest concern to the dentist include the anemias, polycythemia vera, the leukemias, granulocytopenias (agranulocytosis), and the hemorrhagic diseases.

Anemia may range from a state of so-called "chronic deficiency" to a serious blood disorder. In any type of severe anemia there may be pallor of the lips and mucous membranes of the mouth, though, accord-

ing to Anday,[1] "we do not know of any characteristic pathologic change in the gingiva in these cases." In the "aplastic anemias" mouth symptoms are rare, and if they appear, do so at a late stage when the anemia is marked. Acute ulcerative stomatitis and glossitis have been reported in some cases.[31]

Polycythemia vera, a condition in which the red cell count is above normal, is often evidenced in the mouth by a marked bluish-red, cyanotic color of the lips, tongue, and gingivae. Prolonged hemorrhage may accompany surgical trauma.[2]

LEUKEMIA

Both leukemia, in its several forms, and granulocytopenia are always to be viewed as grave blood dyscrasias. In the chronic leukemias (lymphatic or myelogenous) mouth symptoms are rare, and, if present, are mild and appear usually near the end of the disease's course. Spontaneous gingival bleeding may be present as an introductory symptom or may occur at any stage in the disease. Gingival pallor may develop in the late stages and infrequently inflammation or ulcerous processes may be seen in the oral cavity and throat.[3] On the other hand, the earliest symptoms of acute leukemia are oral and very severe in the majority of cases. They may include hemorrhage, purpura, ulceration (frequently of a necrotic character) and stomatitis.[4]

Agranulocytosis, in its acute "fulminating" form, is short and serious. The onset is sudden, usually beginning with a sore throat, toothache, or stomatitis, with ulcers on the tonsils, gingivae, or elsewhere. These lesions are characterized by deep necrosis, extreme pain, and the absence of any inflammatory reaction. The subacute and recurring forms of agranulocytosis are more prolonged and the chronic forms may last for years or decades.[5]

All of the hemorrhagic diseases may, through prolonged gingival bleeding, reveal themselves to the dentist. One group of these diseases is concerned with a decrease in the platelet count (thrombocytopenic purpura) and another (non-thrombocytopenic purpura) with a normal platelet count but some form of capillary damage.[6] Their differential diagnosis and treatment, as with all blood disorders, lie within the field of medicine but the dentist should be aware of the possibility that such sufferers may come first under his attention. He should make himself thoroughly familiar with (oral) pathosis suggestive of hemopathic disorders and the necessity of his cognizance as to what procedures bear heavy contraindication in the presence of these blood dyscrasias cannot be overemphasized.

The blood dyscrasias have been included in this section on hyperplastic tissue change with no intent to infer that such change is necessarily characteristic of them but because they seemed, in the author's opinion, to lend themselves to categorization here. Some few of them, on the basis of the above descriptions, might have been discussed under ulcerative change. This paper would welcome conclusive information regarding the nature of their basic pathosis as it is apparent in the gingivae. The only aim here is to emphasize the fact that hyperplastic change in the gingiva points to a wide variety of etiological agents and if such change, in the light of a thorough history, is even suggestive of something more than local etiology then the practitioner must act accordingly.

When the gingiva, as a portion of the supportive mechanism of the tooth, is secondarily involved through general systemic disease its prognosis must, of course, take a position secondary in importance to the prognosis for the total individual. Any treatment which seems indicated for the gingiva must be subservient to the physician's therapy directed toward the total picture of general well-being, and no periodontal treatment of any nature is in order until the patient is directed into the hands of a physician. Any treatment plan should be discussed in detail with the

physician and made amendable to his suggestions if the plan is to support the patient's best interests.

The prognosis of any gingival disease primarily of systemic origin depends directly upon the prognosis of the systemic pathosis itself, naturally. Local treatment alone is doomed to fail when systemic disease is a predisposing factor and may, even, in certain instances, endanger the life of the patient. The necessity of cooperation between physician and dentist thus becomes evident.

Particular problems are often raised for the dentist when dealing with patients who are under-going certain therapy regimes. The gingivae will exhibit idiosyncrasies to some agents used as drugs and an understanding of the basic considerations involved will help to clarify the problem.

Sodium diphenyl hydantoinate (sodium dilantin) is a compound used in the control of epilepsy. Its use has been pointed out, according to Kimball,[25] as responsible for the production of marked gingival hypertrophy (sic) in a large number of cases. Stern, Eisenbud and Klatell[36] report the following from a study of 50 cases:

1. Fifty-two per cent of the patients showed changes. In only ten per cent, however, was the hypertrophy severe enough to encroach on the clinical crown, and in only one case was there interference with function. It is emphasized therefore that the average reaction is mild; the severe reaction is exceptional.

2. Local irritation such as occlusal trauma, calculus, faulty fillings, etc., acts as an aggravating factor on an already established hypertrophic tendency. The lack of definite correlation is demonstrated by the absence of changes in many of the most neglected mouths and the presence, on the other hand, of advanced changes in many well-cared for mouths.

3. Daily dosage was found to be unrelated. Examination of the figures for duration of dosage, however, showed a considerable longer average period of administration for the more advanced cases (38 mos.) than for those showing minimal changes (17 mos.).

4. Salivary output was studied. Dryness of the mouth was not commonly found to be present, nor could there be demonstrated any consistent diminution in response to paraffin stimulation.

5. Clinical and laboratory examinations (including urinary excretion tests for vitamin B tolerance) failed to reveal any element of dietary deficiency as a basis for the hypertrophy.

6. Clinically, the hypertrophy is exhibited in two forms. The first begins as triangulation of the papillae and proceeds to lobulation with cleft formation at the coalescence of the lobules. The second form begins as a marginal festoon or crescent and develops as a gradual downward encroachment of the roll upon the crown of the tooth.

7. There seem to be no subjective symptoms associated with the increase of tissue. The severity of the average reaction does not justify the psychic trauma which must necessarily accompany the pointing out of this additional physical defect to the already disturbed epileptic patient. For this reason it has been our policy to institute treatment only in the most severe cases, or where the patient himself first calls our attention to the hypertrophy.

Lyons* contends that gingival enlargement in sodium dilantin therapy occurs only in the presence of local irritation and suggests that the enlargement resulting, a hyperplasia, may be due to leakage of the drug into the tissues because of an increased capillary permeability.

The absence of gingival change in cases where inflammation is present would seem to indicate the absence, in these cases, of some unknown factor, perhaps one giving rise to a tissue-tendency toward hyperplasia.

*Lyons, Harry: personal communication.

The prognosis of such cases is considered good, particularly when discontinuance of the drug is practical. Treatment is complicated if it must be exercised in the presence of continued sodium dilantin therapy. However, with conservative procedures alone it has been possible in some cases to achieve a result which is satisfactory aesthetically and functionally for the patient. In extreme cases, however, gingivoplasty may be desirable. The quality of the home care should be extremely high and lasting results should not be anticipated where patients' cooperation is lax or the individual concerned is not in other respects normal.

ALLERGY

The gingiva, as a structure capable of showing allergic change, has been discussed by Healy,[18] Higgins,[20] Straub,[37] Reid,[30] and others. Straub,[38] in a lengthy article, says: "The cephalometric studies of Brodie have demonstrated that the growth patterns of the cranio-facial complex do not change much from birth to maturity, and that the patterns of each Class II, Division I (malocclusion), of protraction of upper incisors wtih retracted mandible or teeth, are laid down very early. In the light of the foregoing study, an allergic tendency as an influencing agent in the growth of the face must be taken into account as an important factor in nasal, sinus, facial and jaw growth. For instance, since poliomyelitis occurring during childhood may change the pattern of growth of the long bones, it is easily conceivable that an allergy might also alter the inherent and hereditary factors in jaw and facial growth and may account for certain dento-facial anomalies." He makes only casual reference to gingival reactions which might suggest allergic manifestations, saying: "In seven, or seventeen per cent, of the allergic patients studied, a pronounced gingivitis was found which could be distinguished from acute inflammation and hypertrophy of the gums as allergic in type. The interproximal tissue was, in many instances, slightly puffy, and had changed from the usual coral pink to a purplish whitish pink. The gingiva had an appearance and texture somewhat similar to the swollen, dampened, whitish mucous membrane of an allergic nose. In many cases, it apparently needed only the addition of bands (orthodontic) to activate the condition."

This reference to distinguishing characteristics of allergic gingivitis on the basis of color and texture may create some perplexity. It has been the author's experience that less disappointment and confusion will arise if no attempt is made, at this stage of our knowledge, to diagnose gingival disease, with respect to differentiation, on a basis of color and tone changes other than to distinguish between changes characterized by arterial hyperemia or venous congestion.

As regards the old question of allergic reactions to dental acrylics, the work of Haag et al.[32] bears serious consideration. They report: "Methyl methacrylate appears to have no very marked irritant action as tested on the skin of man. The irritation found in one subject may have been due to adhesive tape.

"By comparison with the acute toxicity of common industrial solvents, monomeric methyl methacrylate was found to be less toxic than carbon tetrachloride, chloroform, propyl alcohol, ethyl acetate acetal, and dimethyl ethyl phthalate." Dr. Haag investigated only pure monomer because the non-toxicity of the polymer had been previously established by work done in England and Germany.

Undoubtedly allergic reactions may occur in the gingiva. Considering this tissue physiologically and anatomically there is no reason to suppose they may not. Healy's[19] indication that localized gingival eosinophilias with accompanying decrease of polymorphonuclear leucocytes and relative increase of the nongranular white cells (all indicative of allergic reaction) do occur is suggestive. However, Brown[9] and others say that allergy may be present without any increase in the eosinophile count.

Moreover, an eosinophila is indicative of several other conditions, e. g., intestinal parasites, trichinosis, leukemia, or Hodgkins Disease. The question of whether or not (true) gingival changes of an allergic nature do occur, and if so, what form they take and what factors may play a part in the modification of them, probably represents another void in our present knowledge of gingival disease.

The phrase *desquamative tissue change,* used in relation to the term "gingivitis," signifies the existence of a state of gingival inflammation characterized by a peeling or scaling off of living cells of surface epithelium. The normal gingiva, as it is now understood, is covered by a layer of (cornified) cells, a constant exfoliation of the outermost layers of which is a continual phenomenon. In other words all normal gingivae desquamate continually and the process should not be considered as pathological until cells other than the outer, cornified ones have begun to be shed. No entirely satisfactory explanation of keratinization (cornification) seems to have been advanced. Cowdry[11] is of the opinion that the process may have some relationship to the fact that eipthelial cells showing the greatest degree of keratinization lie farthest from the underlying dermis and its rich blood supply. Ziskin, Blackenburg, and Slanetz[43] produced evidence that estrogen controls and is in direct proportion to the degree of cornification of epithelial cells.

Engel, Ray, and Orban,[14] in a study of desquamative gingivitis, report that its pathogenesis is concerned with a loss, reduction, or alteration of the cementing substance of the epithelial cells and the cementing function of the basement membrane. Available evidence suggests that affected tissues have abnormal amounts of collagenase-like factors. As yet these enzymes (sic) have not been demonstrated or identified. They think the sex hormones may be implicated in some way although they could show no definite sequence of relations between estrogen levels and desquamative gingivitis. (They report that some patients have responded clinically to local applications of estrogen).

Since the oral mucous membranes are affected by the sex hormones (as in pregnancy and menstruation) it may be that the severe alterations in desquamative gingivitis are an extension of the same process in which connective tissue cells have a heightened reactivity to changes in sex hormone levels.[15] Further investigation is certainly needed on the pathogenesis of desquamative gingivitis. At the present time this annoying clinical manifestation offers a doubtful prognosis and probably will continue to do so until scientific investigation offers a basis for treatment to replace present methods of empiricism.

GINGIVAL COLOR

The last of the gingival inflammatory states to be discussed here comprises the *pigmented tissue changes,* with special reference to those of metallic origin. When speaking of pigmentation of the gingivae it may be well to differentiate between gingival color, pigmentation normal to certain gingivae, and pigmentation by extrinsic (metallic) agents.

Gingivae vary widely in the color they present. Hill[22] says that "normal gingivae vary in color in different individuals from light to dark shades of pink, but that in all cases the color is uniform throughout." The author agrees that gingivae do vary in color but it has not been evident that the color is uniform throughout. Among others, Thoma,[40] Hayes,[17] Goadby,[16] and Coolidge[10] have discussed pigmentation of the gingiva. The general consensus seems to be that the gingiva receives any (intrinsic) color it may have from two sources: the color of the circulating blood and any melanogenous pigmentation which may be present. In the first instance the gingiva, because of its structural peculiarities, takes on color by virtue of the pigment in the hemoglobin of its circulating blood. Therefore, the color of gingivae *not* characterized by any considerable degree of

melanogenous pigmentation would depend largely upon the state and degree of keratinization of the gingiva, the amount of blood in the peripheral circulation, and the quantity and quality of hemoglobin in this blood. The color of gingivae which do present melanogenous pigmentation would depend not only on that factor of blood and keratinization but in addition on the nature and quantity of the melanogenous pigment in the tissues.

Therefore, it is conceivable that a great many gingivae may be "healthy" with respect to their color, even though they exhibit a wide range of difference in color. Light or pale pink lies at one end of the range-scale of extremes and the author has had evidence of gingivae at the other end of this range-scale whose color is irregularly pink to brown and brownish-white. As a general rule, it would appear that blond individuals tend toward the pale pink gingivae (usually without melanogenous pigmentation) while brunettes tend toward darker colors of pink with the negro and negroid races showing a predominance of melanogenous pigmentation.

Over and above such colorations normal to a given gingiva, the clinician may come in contact with gingival discolorations in individuals who have been treated with therapeutic injections of heavy metals, a practice formally routine in the treatment of syphilis and now not uncommon in certain dermatological disorders.

Thoma[41] reports that pigmentations in the gingiva may be due to toxic substances absorbed in various ways. "Some of these toxic effects are due to medication; others to exposure in factories to fumes and chemicals and dust of various materials. Baum (1934) in an article on occupational diseases of the mouth has tabulated the various metals that may induce changes in the mouth and includes: arsenic, bismuth, mercury, silver, lead, zinc, tin, phosphorus, and antimony."

For the most part, the gingiva will show a marked range of reactions under the influence of heavy metals, and mercury, lead, bismuth, copper, and phosphorus are probably most often discussed. Seemingly lead and bismuth are the most constant regarding the clinical picture they produce, being responsible for a "clearly defined black or blue-black line folowing the crest of the gingiva, the so-called *bismuth line*."[23] This line varies in width, density of color, and distribution. Undoubtedly the gingival manifestation of lead and bismuth is dependent largely on the state of oral sanitation, i. e. metallic deposition is a phenomenon attendant upon certain circulatory disturbances characteristic of inflammation.

The prognosis for teeth whose supporting structures have been affected by heavy metal intake (or contact) is, of course, directly dependent on the amount of damage which has occurred before treatment is begun. The patient should be withdrawn from contact with the offending agent whenever possible. To enhance the prognosis exhaustive efforts must be made to remove all local irritants from the mouth. When the institution of therapy utilizing heavy metals is considered a necessity, all local irritants should be removed before ingestion is begun in order to keep soft tissue reactions to a minimum.

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Letter to the Editor • • • •

Recent publications in dental literature prompt me to write this letter. In using such a medium to express our views, report experimental results, practical experience, etc. to all concerned in the field, it is of utmost importance that any communication is kept on a high ethical level and that the reader is made aware of the differences between fact and fiction—between scientific findings and opinions! In quoting a few examples from the current dental literature, my aim is to make writers as well as readers more critical.

In a recent book on periodontology,[1] there appear two composite illustrations showing the various stages in pocket eradication, the first a periodontal pocket, the second an "infrabony" pocket. These composite illustrations consist each of six photomicrograms. The first picture shows the case before treatment—the next four show different steps in treatment and the sixth picture shows "the result of treatment." However, the sixth picture is a retouched photomicrogram illustrating the end result *expected* by the author. The reader is led to think that these photomicrograms are documentary evidence to support the author's views. There is no mention anywhere in the book that these photomicrograms have been retouched to illustrate the author's hopes. The technique shown in these pictures is discussed in detail in the text. There are numerous diagrams illustrating the procedure, so that there is no justification for illustrating the same thing by seemingly "documentary" evidence.

An excellent scientific paper on the epithelial attachment appeared in the April 1953 issue of the Journal of Periodontology.[2] The author describes a new method of studying the structure of the epithelial attachment by phase contrast microscopy. As far as scientific planning and execution are concerned, this paper can be considered a very fine example of research in the field of dental histology. However, an author of high standing should be more careful in his manner of handling the literature. Every scientific worker has to be cognizant of what has been done in his field of investigation by his predecessors. This is of importance not only from the standpoint of giving due credit where it should be given (often the only real compensation to a research worker) but to avoid unnecessary duplication of work. The statement of the author on the tonofibrils, as follows: "Because they disintegrate soon after cell death and are only faintly preserved by fixing methods, they heretofore escaped the attention of observers" does not take into consideration former observations. The presence of tonofibrils was established long before phase microscopy was developed. Attention was focused on the tonofibrils in connection with the problem of attachment of epithelium to the root surface in a paper published in 1948[3] and even earlier in the German literature.

Another statement in this article: "The relatively late formation of the primary cuticle, as well as the protracted presence of ameloblasts such as recently disclosed[3] . . . . ."[4] is inaccurate. In the book on "Biology and Pathology of the Tooth and Its Supporting Mechanism,"[5] one may read the following sentence on Page 8: "When the summit of the crown erupts into the oral cavity, the root end is still incompletely formed; even the enamel, in many instances, may be in the process of formation in the cervical region of the crown as indicated by the presence of occasional ameloblasts." This fact has also been stated as follows on the gingival pocket in the German literature:[6] "Oft finden wir bei bereits durchgebrochenen Zähnen die Ganoblasten (ameloblasts) in der Nähe des Schmelzendes, ihrer jungsten Bildungsstätte, noch erhalten, während Naher zum Taschenboden die Ganoblasten sich bereits zurückgebildet haben."

I would like to stress the fact that such inaccuracies are frequent in dental literature. I selected the quoted paper for criticism because I consider it in all other respects as a paper of high scientfic value.

Two reviews, of Waerhaug's book, "The Gingival Pocket" have recently been published.[7] Both demand our attention.

There is a difference between abstracting and critically discussing a book. However, the reviewer who expresses his own opinion of a book must realize that he exposes himself to criticism.

One reviewer states that "Waerhaug indicates with a great deal of *convincing evidence* that after eruption, the epithelium is nowhere organically attached to the enamel . . ." On the same subject, the other reviewer expresses himself as follows: "Dr. Waerhaug *satisfactorily* establishes the general conclusion that a physiologic pocket extending to the cemento-enamel junction is formed upon the eruption of the tooth." The reviewers identify themselves with Waerhaug's conclusion by using the term "convincing evidence" and "satisfactorily." To me, Waerhaug's "evidence" on the nature of the epithelial attachment is neither convincing nor satisfactory.

Conclusions of such far-reaching consequences cannot be accepted without carefully checking the methods used by the author, by confronting them with old evidence, and if possible, by testing them with new evidence and findings. The immediate practical application of Waerhaug's findings can only be judged precocious.

Statements such as the following have to be sharply refuted: "The routine use of resection to eliminate pockets and reduce them to zero has therefore no longer any good theoretical basis," and "It clarifies the theory underlying our attempt to get reattachment and shows that while we do not get organic reattachment, we do get sealing of pockets and sulci when our treatments are successful." Such statements by a reviewer indicate more wishful thinking than respect for scientific evidence and sound clinical practice.

On the other hand, it seems not to be good practice to refer cursorily in a review to one's own unpublished findings as if they had been amply established, to quote as follows: "My own observation does not tally with that of Waerhaug in his findings that stress has little or nothing to do with pocket deepening. My own findings indicate that stress which causes lateral movements of the tooth in its socket is an important cause of inflammatory effects which act much like those due to deposits and may indeed invite penetrating bacterial infection." Such a statement *calls for immediate publication of relevant data.*

BALINT ORBAN.

Colorado Springs, Colorado—July, 1953

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Reviewed by: Dr. Harold J. Leonard, J. Period., April, 1953; Dr. G. R. Lundquist, J.A.D.A., June, 1953.

Announcements

REPORT OF MEETINGS OF TEACHERS OF PERIODONTOLOGY HELD IN ENGLAND

Growing appreciation of the importance of the supporting tissues of the teeth has been manifested by an increase in the number of appointments in Periodontology which have been made in British Dental Schools during the past few years.

The need for co-ordination and the achievement of a certain degree of uniformity in teaching has long been felt, and at the instigation of the British Society of Periodontology a meeting of those engaged in teaching this subject was held in London in December, 1952.

A further short meeting was held at Liverpool in February, 1953, at the time of a meeting of the British Society of Periodontology, when it was agreed that the first problem to be discussed was that of terminology. This was done at Buxton on 6th and 7th July, 1953, in conjunction with the Annual Meeting of the British Dental Association which was devoted to an appraisal of periodontal diseases.

Appended to this report are the recommendations of the teachers of periodontology, who will meet again next year to discuss the classification of periodontal diseases. The recommendations arose from a consideration of the definitions approved by the 1949 Nomenclature Committee of the American Academy of Periodontology, a Society which has done invaluable work on Nomenclature and Terminology.

These recommendations are being communicated to the Association pour les Recherches sur les Paradentopathies (Arpa Internationale) and the American Academy of Periodontology in the hope that a greater degree of mutual understanding at an international level will result.

NOMENCLATURE RECOMMENDATIONS OF THE TEACHERS OF PERIODONTOLOGY OF GREAT BRITAIN AND EIRE (1953)

Alveolar Crest: The free border of the alveolar process.

Alveolar Process: That portion of the jaw bones which supports the teeth, develops with them, and largely disappears when they are lost.

Alveolus, Dental: The tooth socket.

Bruxism: Grinding or clenching of the teeth in other than normal function.

Crevicular Epithelium: The epithelial lining of the gingival crevice.

Dental Calculus: Deposits on the teeth consisting of inorganic salts deposited into an organic matrix. It may be either supra- or sub-gingival or both.

Epithelial Attachment: The epithelium which is attached to the tooth surface for a variable distance apically from the base of the gingival crevice or periodontal pocket.

Epulis: A localized swelling of the gingiva.

Gingiva: That part of the oral mucous membrane which is firmly attached to the alveolar processes and surrounds the teeth.

Gingival Crevice: (Gingival Sulcus, Gingival Trough): The shallow crevice between the tooth and the free gingiva.

Gingival Hyperplasia: An enlargement of the gingiva resulting principally from a numerical increase of the tissue elements.

Gingival Recession: A progressive exposure of the root surfaces by apical shift of the gingiva, associated with resorption of the alveolar crest and loss of the corresponding part of the periodontal membrane.

Gingivitis: Inflammation of the gingiva.

Lamina Dura: The bone lining the tooth socket. (This is not considered an entirely suitable term).

Oral Mucosa (Oral Mucous Membrane): The lining of the oral cavity.

Periodontal Abscess: A localized collection of pus originating within the periodontal tissues.

Periodontal Pocket: A pathologically deepened gingival crevice.

1. *True:* One in which destruction of the supporting tissues and apical proliferation of the epithelial attachment has occurred.
   a. *Intra-Bony:* One in which the base is apical to the alveolar crest.
2. *False:* One in which no destruction of the supporting tissues and no apical proliferation of the epithelial attachment has occurred.

Periodontal Membrane: The tissue which lies between the cementum and the surrounding tissues.

Periodontal Tissues or Periodontium: The investing and supporting tissues of the tooth, namely, the gingiva, periodontal membrane, cementum and alveolar process.

Periodontology: The science and study of periodontal diseases.

Periodontitis: Inflammatory involvement of the periodontal tissues which is not limited to the gingiva.

Vincent's Disease: A type of acute or sub-acute ulcero-membranous gingivo-stomatitis.

The following terms were considered to be unnecessary:—
Bruxomania, periodontia, periodontics, periodontist and traumatism.

It was considered unnecessary to define "periodontal diseases."

Periodontal atrophy was considered, but not defined.

A. Bryan Wade, Hon. Secretary. W. G. Cross, Chairman.

WILLIAM J. GIES FOUNDATION

The William J. Gies Foundation for the Advancement of Dentistry, Inc., has been named a beneficiary in the will of the late Dr. Henry Spenadel of New York City. The will provides that the amount of $500 shall be paid each year to the Foundation in perpetuity, to be used for the general and specific objects of the income fund as the Foundation may deem fit. It is stipulated, however, that this annual income shall not be added to, or become part of a capital fund of the Foundation.

It is estimated that the minimum income of the Foundation from this bequest and from its other funds will be between $1300 and $1400 annually.

The Foundation is currently organizing, on a national scale, a committee of friends and admirers of Dr. Gies for the purpose of enlarging the capital fund of the Foundation so as to increase its usefulness in the advancement of dentistry.

The principal avenues through which the Foundation seeks to advance its objectives are in the fields of dental research, dental journalism and literature, dental idealism and all those intangibles which together elevate the status and effectiveness of dentistry.

AMERICAN ACADEMY OF DENTAL MEDICINE ANNOUNCES MID-WINTER MEETING

The American Academy of Dental Medicine will hold its Mid-Winter Meeting and Luncheon on Sunday, December 6, 1953, at the Hotel Statler, N. Y. C. "Geriatrics" will be the theme of the scientific session. There will be a business meeting for members at 10 A.M. The Luncheon and Round Table Discussions on geriatrics and related subjects will begin at 12:30 P.M.

All interested dentists and physicians are cordially invited to attend. For details and reservations address the National Secretary, Dr. William M. Greenhut, 124 East 84th Street, New York 28, New York.

The establishment of two Fellowships for clinical and experimental research has been announced by the Trustees of the Jewish Sanitarium and Hospital for Chronic Diseases. The Fellowships, named in the honor of the President of the Hospital, Mr. Isaac Albert, will be awarded during the fall of 1953.

Applicants should write to the Hospital Superintendent, Rutland Road and East 49th Street, Brooklyn, New York.

PERIODONTICS

Jacoby T. Rothner, D.D.S., F.A.C.D., Professor of Periodontics
Temple University School of Dentistry and Staff

To give the general practitioner the most recent developments and changed concepts in the modern practice of periodontics—this is our object.

The course will start with considerations of the normal tissues as a basis, and go on to the changes which may be manifested. The approach to the analysis of the causes, diagnosis, and treatment of the diseases of the supporting tissues will be of a practical nature. There will be laboratory practice and clinical demonstration in occlusion, instrumentation, and home-care.

Applications for enrollment in this course should be sent to: Dr. Louis Herman, Temple University School of Dentistry, 3223 North Broad Street, Philadelphia 40, Pennsylvania.

CHICAGO COLLEGE OF DENTAL SURGERY

The Chicago College of Dental Surgery, Loyola University, anounces a 3 day course on Theory and Practice of Periodontics. The course will be conducted by Professor Balint Orban and Staff and will consist of seminars and clinical work. The dates are January 21, 22, 23, 1954. Those wishing to apply may contact Dr. F. M. Wentz, Director, Postgraduate Division, 1757 West Harrison Street, Chicago, Illinois.

NEW YORK UNIVERSITY COLLEGE OF DENTISTRY

Postgraduate Courses Starting in October

Course	*Dates*	*Day*	*Hour*
Periodontia	10/ 7/53-12/ 9/53	Wed.	7-9
Pathology	10/ 9/53-12/18/53	Friday	1-4
Airbrasive	10/12/53-10/17/53	one week	
Anatomy	10/14/53-/12 2/53	Wed.	7:30-10
Roentgenology	10/15/53-11/12/53	Thurs.	4-6
Operative Dentistry	10/16/53- 2/12/54	Friday	4-7

For further information or application write to: Miss Catherine Iozzo, Secretary, Postgraduate Division, New York University College of Dentistry, 209 East 23rd Street, New York 10, New York.

SCHOOL OF DENTISTRY, UNIVERSITY OF PENNSYLVANIA POSTGRADUATE COURSES

P.G. 22—by Henry M. Goldman, D.M.D., and Associates—Periodontal Therapy—14 to 19 December 1953

The objective of this course is to acquaint the practitioner with the importance of periodontics in the general practice of dentistry. The etiology and diagnosis of periodontal diseases will be stressed. The various technics of periodontal therapy will be discussed from a point of view of indications, contra-indications and methods. Case planning will be emphasized. This course includes lectures and small group demonstrations.

P.G. 24—by Ernest R. Granger, D.D.S.—Precision Prosthesis and Functional Treatment—18 to 23 January 1954

This course on partial dentures will deal with the use of precision prosthesis as a means of restoring and maintaining the function of its opposing, apposing, supporting structures.

For further information write to: Postgraduate Courses, School of Dentistry, University of Pennsylvania, 4001 Spruce St., Philadelphia 4, Pa.

COLUMBIA UNIVERSITY

A course on the Principles of Occlusion, PD 429, consisting of lectures, seminar conference and group participation, and clinical demonstrations will be given by Professor Lewis Fox and associates on January 13, 14, and 15, 1954, at Columbia University School of Dental and Oral Surgery.

The fee is $150. For further information write to Dr. Barnet M. Levy, Director, Postgraduate Division, School of Dental and Oral Surgery, Columbia University, 630 West 168th Street, New York 32, N. Y.

INDEX

The Journal of Periodontology, Volume 24, 1953

JANUARY

Systemic Aspects of Periodontal Disease	7	Lester W. Burket
The Attachment of Calculus to Root Surfaces	16	Helmut A. Zander
The Effect of Toothbrushing on the Keratinization of the Gingiva	20	S. S. Stahl Nathan Wachtel Carlos De Castro George Pelletier
Biopsy Studies of Necrotizing Ulcerative Gingivitis	22	Erwin M. Schaffer
Reattachment in Periodontal Disease	26	Max Shapiro
White Folded Gingivo-Stomatosis	32	Frank G. Everett Harold J. Noyes
The Apparent Antagonism Between Caries and Periodontal Disease	42	Basil G. Bibby
Book Review	53	
Editorials	54	
Announcements	58	

APRIL

Dr. Grace Rogers Spalding: In Memoriam	70	
The Crest of the Interdental Alveolar Septa	75	Beryl Ritchey Balint Orban
A Rational Plan for Periodontal Therapy	88	Sigurd Ramfjord
A Calibrated Silver Point for Periodontal Diagnosis and Recording	94	Leonard Hirschfeld
The Structure of the Epithelial Attachment	99	Louis J. Baume
Attachment Level Patterns of Interproximal Pockets	111	Melvin L. Morris
Thiamin Hydrochloride as an Adjunct in Surgical Periodontal Therapy	116	Bernard Herzog John O. McCall
Gingival Manifestations of Lupus Erythematosus	119	Theodore Lite
American Board of Periodontology	123	Harold J. Leonard
Book Review: The Gingival Pocket by Jens Waerhaug	124	Harold J. Leonard
Announcements	87, 122, and 128	

JULY

The Effects of Various Levels of Dietary Protein on the Periodontal Tissues of Young Rats	135	Asger M. Frandsen, et al.
Clinical Manifestations of Periodontal Disturbances Requiring Treatment	143	Edgar D. Coolidge
Psoriasis Gingivae	156	H. A. Brayshaw, et. al.
The Effect of the Systemic Administration of Cortisone Upon the Periodontium of White Mice	161	Irving Glickman, et al.
Pre-Operative Procedures and Post-Operative Expedipents in Periodontal Surgery	167	Raoul H. Blanquie
Tissue Reactions Around Artificial Crowns	172	Jens Waerhaug
Case Report: Abrasion Through the Abuse of Dental Floss	186	Frank G. Everett, et al.
Results of a Three Year Study of Vincent's Infection at the Great Lakes Naval Dental Department	187	W. J. Carter, et al.
Announcements	195	

OCTOBER

Gingivosis	207	Calvin L. Foss, et al.
The Reattachment of Human Periodontal Tissues Following Surgical Detachment: A Clinical and Histological Study	220	Melvin L. Morris
Temporary Splinting An Adjunct in Periodontal Therapy	229	Nathan Friedman
The Dentin: Its Physical Characteristics During Curettage	232	A. B. Riffle
The Etiology of Periodontal Disease	242	Paul E. Boyle
Report of a Study on the Relationship of Personality Variables to Periodontal Conditions	248	John H. Manhold
An Evaluation of Factors Determining Prognosis in Inflammatory and Retrogressive Periodontal Disease: A Series	252	Richard William Slatten
Letter to the Editor	265	Balint Orban
Announcements	251-267	

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